# Cytomegalovirus and Transmission via Breast Milk

## *How to Support Breast Milk to Premature Infants and Prevent Severe Infection?*

***To the Editors:***

Recently, we published a review on transmission of human cytomegalovirus (HCMV) via breast milk to the premature infant.[1] Studies revealed HCMV positivity of the infants of mothers being HCMV-IgG positive ranging from 5.7% to 58.6%, with symptomatic HCMV disease occurring in 0%–34.5% (median, 3.7%) and severe sepsis-like syndrome in 0%–13.8% (median, 0.7%). Evidence for long-term sequelae was scarce, suggesting only mild neurologic and cognitive impairment without hearing impairment. Recently, Hamele et al[2] reported on severe morbidity and mortality associated with breast milk-acquired HCMV infection in 5 preterm infants of 24 (+5) to 27 (+1) weeks gestational age. These 5 cases add to a total of 18 infants that have been published since the early 1970s when human breast milk was known for the first time to be a potential source of HCMV infection. In 2 cases of these 18 infants, no details were provided, another 5 cases of 29 to 33 weeks gestational age did not experience severe disease as defined as sepsis-like syndrome, and the remaining 11 infants from 4 studies were reported to range from 23 to 28 weeks (23, 25, 24–28, and 24.4 ± 0.5 weeks, respectively).

Pasteurization constitutes the preferred procedure to inactivate HCMV in breast milk because freezing does not completely eliminate the virus.[3] However, heating procedures significantly decrease protective factors contained in breast milk, which are fundamental for the advantages and possible long-term protection granted by breast-feeding.[4] Despite the concerns over severe HCMV disease in a minority of very low birth weight infants, we would prefer an individual decision based on the health status of the preterm infant instead of a general approach by either pasteurization or withholding of breast milk.

We believe that parents should be informed about the risk of HCMV acquisition of their preterm infant and the advantages of fresh milk feeding. Provided that informed consent is given and the preterm infant is in stable condition, fresh breast milk might be administered (Fig. 1). The suggested algorithm includes a careful risk-to-benefit evaluation of the infant's current health status and the benefits of its own mother's breast milk that might, in our opinion, clearly outweigh the risk of symptomatic HCMV infection. Weekly HCMV monitoring in the infant by polymerase chain reaction from urine samples is mandatory, and if the infant becomes positive, feeding fresh milk must be stopped to reduce viral loads in case of symptoms and signs of HCMV infection. Otherwise, feeding of fresh breast milk might be continued in infants of >28 weeks of gestational age. At a corrected age of >32 weeks, the physician could stop pasteurization or withholding of breast milk, based on the infant's health status and a more mature immune system. We still share the view of Stagno et al[5] that the low risk associated with the transmission of HCMV through breast milk is clearly outweighed by the well-established value of breast-feeding. Passive immunization with either HCMV monoclonal antibodies or immune globulins might be a case of debate for high risk low birth weight infants.

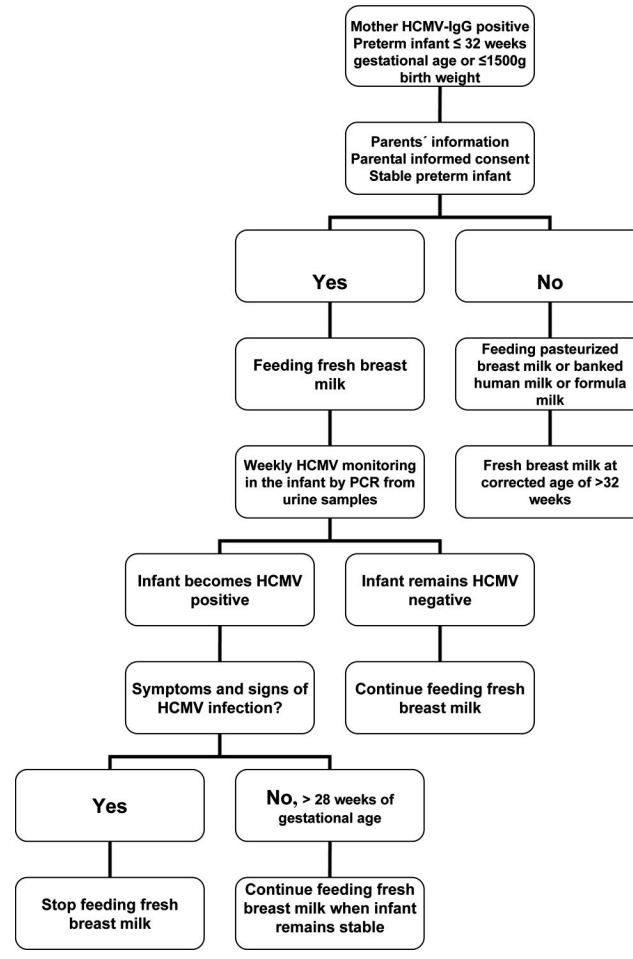


**FIGURE 1.** A new algorithm suggested for breast-fed preterm infants <32 weeks of gestational age or birth weight below 1500 g in case of HCMV-IgG–positive mother.

**Stefan Kurath, MD**
**Bernhard Resch, MD**
Research Unit for Neonatal Infectious Diseases and Epidemiology
Division of Neonatology
Department of Paediatrics
Medical University of Graz
Graz, Austria
bernhard.resch@medunigraz.at

## REFERENCES

1. Kurath S, Halwachs-Baumann G, Müller W, et al. Transmission of cytomegalovirus via breast milk to the premature born infant: a systematic review. *Clin Microbiol Infect.* 2009 [Epub ahead of print].

2. Hamele M, Flanagan R, Loomins A, et al. Severe morbidity and mortality with breast milk associated cytomegalovirus infection. *Pediatr Infect Dis J*. 2010;29:84–86.
3. Hamprecht K, Maschmann J, Müller D, et al. Cytomegalovirus (CMV) inactivation in breast milk: reassessment of pasteurization and freeze-thawing. *Pediatr Res*. 2004;56:529–535.
4. American Academy of Pediatrics, Workgroup on Breastfeeding. Breastfeeding and the use of human milk (RE9729). *Pediatrics*. 1997;100:1035–1039.
5. Stagno S, Reynolds DW, Pass RF, et al. Breast milk and the risk of cytomegalovirus infection. *N Engl J Med*. 1980;19:1073–1076.

# Update on Meningococcal Disease Mortality in the United States Since 2002

***To the Editors:***

Since the publication by Sharip et al[1] on national meningococcal disease mortality (1990–2002), there has been an absence of further analysis of mortality trends of this disease in the United States. The present communication provides an update of US meningococcal disease mortality trends, based on an analysis of multiple cause-of-death data[2] between 2002 and 2006.

During 2002 to 2006, the age-adjusted average annual meningococcal disease mortality rate was 0.050 per 100,000 person-years (95% confidence interval [CI], 0.047–0.054). Individual annual rates decreased by 34.5% from 0.058 per 100,000 person-years (95% CI: 0.049–0.067) in 2002 to 0.038 per 100,000 person-years (95% CI: 0.031–0.045) in 2006. Poisson regression analysis showed that during this time the average annual decline was 10.3%.

Four age groups experienced significantly higher average annual mortality rates than both the general population and the non-Hispanic white racial/ethnic group (whites) between 2002 and 2006. Infants (age <1 year) had the highest mortality rate (0.33/100,000 person-years), followed by the age groups: 85+ years (0.13/100,000 person-years), 1 to 4 years (0.10/100,000 person-years), and 15 to 24 years (0.076/100,000 person-years). Among all racial/ethnic groups, non-Hispanic blacks (blacks) experienced the highest age-adjusted average annual meningococcal disease mortality rate of 0.069 per 100,000 person-years (95% CI: 0.057–0.081). The black racial/ethnic group had an age-adjusted rate ratio of 1.39 (95% CI: 1.24–1.55), when compared with whites.

In comparison with the 1990–2002 analyses by Sharip et al,[1] the age-adjusted average annual meningococcal disease mortality rate during 2002–2006 was significantly lower; however, group disparities continued to persist. The meningococcal disease mortality burden remained substantial for blacks, infants, children aged 1 to 4 years, adolescents and young adults aged 15 to 24 years, and adults older than 85 years.

Although these observed declines in mortality rates may in part reflect the effectiveness of recent public health control and prevention efforts, it is still disconcerting to see that children aged 1 to 4 years and young adults aged 15 to 24 years are continuing to experience higher rates of meningococcal death than the general population, especially given the availability of a tetravalent meningococcal conjugate vaccine (MCV4) since 2005. The MCV4 is presently approved for use among children and adults aged 2 to 55 years.[3]

The present multiple cause-of-death analysis underscores the continual need for longitudinal evaluations of meningococcal disease trends and the effects of MCV4 on decreasing disease outbreaks and mortality and on increasing herd immunity in the future. More vigilant surveillance and timely analysis of meningococcal disease morbidity and mortality may provide further insights into best practices for effective prevention and control of this potentially vaccine-preventable disease.[4]

The findings also suggest that physicians should target high-risk children and adolescents, and educate these patients and their parents about the availability and potential benefits of the MCV4 vaccine.[4] Plausible policy strategies that can further reduce the burden of meningococcal disease in the United States include publicly funded vaccination of high-risk groups, mandated use of conjugate vaccines as part of routine well child care, and increased access to affordable vaccines in disadvantaged communities.[4]

**Gloria Y. Kim, MPH**
Los Angeles County Department of Public Health

**Frank Sorvillo, PhD**
Department of Epidemiology
UCLA School of Public Health

**Tony Kuo, MD, MSHS**
Los Angeles County Department of Public Health
Department of Family Medicine
David Geffen School of Medicine at UCLA
Los Angeles, CA

## REFERENCES

1. Sharip A, Sorvillo F, Redelings MD, et al. Population-based analysis of meningococcal disease mortality in the United States. *Pediatr Infect Dis J*. 2006;25:191–194.
2. Redelings MD, Wise M, Sorvillo F. Using multiple cause-of-death data to investigate associations and causality between conditions listed on the death certificate. *Am J Epidemiol*. 2007;166:104–108.
3. American Academy of Pediatrics Committee on Infectious Diseases. Prevention and control of meningococcal disease: recommendations for use of meningococcal vaccines in pediatric patients. *Pediatrics*. 2005;116:496–505.
4. Moore KA, Osterholm MT. Meningococcal disease and public health: a complicated road map. *JAMA*. 1998;279:472–473.

# Chemotherapy and Surgery in Children With Cystic Echinococcosis

***To the Editors:***

Cystic echinococcosis (CE), caused by *Echinococcus granulosus*, causes a long-lasting infection that affects humans and a wide range of livestock species.[1] In the human host, cysts can develop in many anatomic sites, especially liver and lung. Because of the slow cyst growth, diagnosis of CE in children with multiorgan involvement is rare, even in endemic countries; the treatment strategy in CE remains an open question.[2]

We describe a case observed in September 2003 of a 9-year-old boy, living in a farm in the south of Italy, who was admitted to our hospital because of persistent cough and expectoration of bloody sputum. Thoracoabdominal computed tomography scans showed a large cyst (4 cm diameter) in the mediastinum near the left ventricle, multiple cysts in the lung, a cyst in the liver, and a very large cyst (8 cm diameter) in the pancreas; echocardiography showed an intrapericardial cyst (2 cm diameter). Nuclear magnetic resonance confirmed the CE diagnosis. Laboratory tests showed eosinophilia of 17%, anti-*Echinococcus* antibody seropositivity (hemagglutination test: antibody titer: 1:16,384), immunoblotting: antibodies against 8, 16, 20 kd (antigen B), and 55 to 65 kd (antigen 5) antigenic subunits.

Treatment was started with albendazole (10 mg/kg/d). One month after the start of anthelminthic therapy, an echocardiography showed a volumetric increase of the pericardial cyst (3 cm diameter). After 45 days of chemotherapy, complete surgical resection of heart cyst and concomitant cleaning of liver and lung cysts became necessary. The patient underwent elective operation in 1-stage surgery, through a median sternotomy incision and next xiphoum-

bilical incision. The cyst mass was seen clearly on the wall of the left atrium and completely dissected away. Lung cysts of left superior and right inferior lobes and the liver cyst of left lobe were removed. Finally, the peritoneum was raised and the cyst located in the pancreas was completely removed. Histologic examination of the surgical material confirmed the diagnosis of CE. The patient was extubated on second postoperative day and 1 week later discharged home with continuous albendazole therapy for 2 years (10 mg/kg/d). The postoperative period was uneventful.

Four years' clinical, serologic, and ultrasonographic follow-up has shown no recurrence. To note, although, enzyme-linked immunosorbent assays determining isotype antibody expression in response to *E. granulosus* hydatid fluid, showed no significant variations for total immunoglobulin (Ig) G and IgG1 concentrations, IgG4 and IgE concentrations dropped at completion of follow-up (IgG4:O.D.: 1.22 vs. 0.64; IgE: O.D.:0.18 vs. 0.008).

CE is endemic in Italy, in particular in the south of the country. Because the cyst requires many years to develop, the diagnosis of pediatric echinococcosis is rare, and the multiorgan localization in child is uncommon.[3,4] In particular, cardiac and pancreatic involvements, which occur only after the larvae pass trough the barrier of liver and lung, are rare in children (0.2%–2%). In a Turkish study, a 13-year-old girl presented hydatid cyst of the right atrium, cyst in the kidney, and multiple cyst in the lungs.[5] This is the first report of a child with hydatid with a cyst in the hearth, pancreas, lung, and liver. Although operating on 4 distinct organs is a high-risk procedure, we decided for a 1-stage surgery because we did not want to risk spreading the infection and the fatal complication from the growth and/or rupture of the cardiac cyst.

This noteworthy case of a young patient with multiorgan involvement shows that combined chemotherapy and surgery allow to successfully treating severe CE.

**Maria Iannelli, MD**
Department of Pediatric Medicine (DMP)
Pediatric Hospital Bambino Gesù, IRCCS

**Alessandro Inserra, MD**
**Roberto M. Di Donato, MD**
Pediatric Hospital Bambino Gesù, IRCCS

**Antonella Teggi, MD**
Department of Infectious Diseases
Hospital Sant'Andrea
University of Rome, "Sapienza"

**Alessandra Siracusano, PhD**
Department of Infectious Parasitic and
Immune-mediated Diseases
Istituto Superiore di Sanità

**Saverio Malena, MD**
Pediatric Hospital Bambino Gesù, IRCCS

**Renata Boldrini, MD**
Pathology Unit-Laboratory Department (DL)
Pediatric Hospital Bambino Gesù, IRCCS

**Cristina Russo, MD**
Pediatric Hospital Bambino Gesù, IRCCS

**Andrea de Zorzi, MD**
Department of Pediatric Cardiology and
Cardiac Surgery (DMCCP)
Pediatric Hospital Bambino Gesù, IRCCS

**Alberto Villani, MD, PhD**
Department of Pediatric Medicine (DMP)
Pediatric Hospital Bambino Gesù, IRCCS
Rome, Italy

## REFERENCES

1. Eckert J, Deplazes P. Biological, epidemiological, and clinical aspects of echinococcosis, a zoonosis of increasing concern. *Clin Microbiol Rev*. 2004;17:107–135.
2. Vuitton DA, Wen H. Treatment of cystic echinococcosis: a combination of general goals and rules, individual decisions and indications. *Neth J Med*. 2007;65:86–88.
3. Emilio C, Losurdo G, Mollero L, et al. Multiorgan echinococcosis in a pediatric patient. *Pediatr Infect Dis J*. 2005;24:660–661.
4. Olmez D, Babayigit A, Arslan H, et al. Multiorgan involvement in a pediatric patient with hydatid disease. *J Trop Pediatr*. 2008;54:417–419.
5. Aydoğdu T, Sahin N, Ulusan V, et al. Right atrial hydatid cyst associated with multiple organ involvement: case report. *J Thorac Cardiovasc Surg*. 2001;121:1009–1011.

# Prevalence of Scabies and Head Lice Among Students of Secondary Boarding Schools in Kuching, Sarawak, Malaysia

***To the Editors:***

We embarked on a study to determine the prevalence of scabies and head lice among secondary school students attending boarding schools in Kuching, Sarawak, because we had noted that most of our clinic patients with infestations were boarding school students. The aim was to provide these data to the authorities to formulate a strategy to eliminate these infestations from boarding schools in Sarawak.

We randomly selected 2 of 9 boarding schools in Kuching, Sarawak, an Islamic school and a technical school. The protocol for this cross-sectional study was approved by the school authorities and the State Health and Education Department. The study was conducted between March and May 2009. All students who voluntarily consented were surveyed. The diagnoses were made clinically. Mass treatment of all the students was conducted after the survey. Data were subjected to descriptive analysis.

A total of 944 of 950 students consented to the survey. Of these, 488 (51.7%) were males and 456 (48.3%) were females. The median age was 16 years, ranging from 13 to 17. Malays constituted the majority with 708 (75.0%), followed by Bidayuhs 121 (12.8%) and Ibans 65 (6.9%).

We found that 233 (24.7%) students had head lice, and 76 (8.1%) students had scabies. All the students with head lice were females. This constituted 48.9% of the total female students surveyed. Scabies were seen in 61 males and 15 females. These constituted 12.5% of the male students and 3.3% of the female students studied. The point prevalences for head lice and scabies were 40% and 10.4% in the Islamic school and 7.1% and 5.7% in the technical school, respectively.

Our point prevalence for head lice of 24.7% was twice that for the prevalence of 12.9% among primary school children in Kuala Lumpur, Malaysia.[1] The higher rate might have resulted from the fact that the survey was done in boarding schools where students were in close contacts with each others in the classes and in the dormitories.

We found that only female students were infested with head lice. Interestingly, we also found that the infestation by head lice was more common in the Islamic school than in the technical school. We suspect that the prevalence of head lice was higher in Muslim females because of the practice of wearing a head scarf, keeping long hair, infrequent hair washing, and the proximity among these students during mass prayers. Moreover, the dormitories in the Islamic schools were more overcrowded, housing 12 students per room compared with 8 in the technical school. In Nigeria, it was also found that the infestation rate was higher in the Islamic community with 4.1% compared with the Christian community rate of 3%.[2] Females, longer hair, less frequent hair washing, sharing cleaning implements, and overcrowding were also identified as risk factors for head lice infestation.[3,4]

Scabies was found in 8.1% of the students in our study. In a survey among primary school children, the prevalence was 4% in urban Mali, 0.7% in Malawi, and 4.3% in rural Cambodia.[5] The higher prevalence seen here might be due to the proximity and frequent handshaking among Muslim students in the boarding schools.

In conclusion, the prevalence of these infestations is high, warranting mass treatment by the education and health authorities to contain the transmission in all the boarding schools in Sarawak. Control of these infestations is important to allow a better quality of life and improve the educational and cocurricular performance of the boarding school students. We recommend that school authorities screen all new students for these infestations and refer them for appropriate treatment before starting school to prevent transmission of these diseases.

**Felix Boon-Bin Yap, MD, MRCP**
**Estrellita M. T. Elena, MB BS**
**Muniandy Pubalan, MB BS, MRCP**
Department of Dermatology
Sarawak General Hospital
Sarawak, Malaysia

## REFERENCES

1. Sinniah B, Sinniah D, Rajeswari B. Epidemiology and control of human head louse in Malaysia. *Trop Geogr Med*. 1983;35:337–342.
2. Ebomoyi EW. Pediculosis capitis among urban school children in Ilorin, Nigeria. *J Natl Med Assoc*. 1994;86:861–864.
3. Counahan M, Andrews R, Büttner P, et al. Head lice prevalence in primary schools in Victoria, Australia. *J Paediatr Child Health*. 2004;40:616–619.
4. Ríos SM, Fernández JA, Rivas F, et al. [Pediculosis prevalence and associated risk factors in a nursery school, Bogota, Colombia] [Article in Spanish]. *Biomedica*. 2008;28:245–251.
5. Landwehr D, Keita SM, Pönnighaus JM, et al. Epidemiologic aspects of scabies in Mali, Malawi, and Cambodia. *Int J Dermatol*. 1998;37:588–590.

# Lack of Sensitivity of QuantiFERON-TB Gold Test in Tube in a Child With Tuberculous Meningitis

***To the Editors:***

Diagnosis of tuberculous meningitis remains an important clinical challenge. About half of patients will present with normal chest radiography or a negative tuberculin skin test (TST). Negative smears for acid-fast bacilli and lack of isolation of *Mycobacterium tuberculosis* in cerebral spinal fluid (CSF) culture are observed in more than 50% of cases. The CSF polymerase chain reaction (PCR) assay represents a diagnostic advance, but is insensitive to confidently exclude the diagnosis. Recently, interferon-gamma release assays (IGRAS) have been reported to improve diagnostic sensitivity of tuberculosis in adults and children, but studies in tuberculous meningitis are lacking.

We have recently diagnosed a tuberculous meningitis in a 2-year-old Rumanian girl who presented with a negative TST (0 mm), normal chest radiograph, negative CSF smears for acid-fast bacilli, negative CSF-PCR assay for *M. tuberculosis* and negative QuantiFERON-TB Gold Test In Tube (QTF) (0.27 UI/mL; positive value $\geq$0.35 UI/mL) with proper interferon production in mitogen control (6.21 UI/mL). At time of admission, CSF had a mildly elevated pleocytosis (60 leukocytes/mm$^3$; 85% neutrophils), a slight raised protein (90.7 mg/dL) and a low glucose value (14 mg/dL) and computed tomography scan revealed mild basilar meningeal enhancement. There was no evidence of tuberculosis exposure. One month after admission, *M. tuberculosis* was isolated in CSF and gastric aspirate contents. A new QTF and TST were again performed and were positive (0.72 UI/mL and 13 mm, respectively).

The few available data of the use of IGRAS for diagnosing extrapulmonary tuberculosis suggest that these assays have the same sensitivity as in pulmonary disease.[1]Regarding the use of QFT for tuberculous meningitis diagnosis, few articles have been published.[2,3] All of them estimate that QTF sensitivity is higher than acid-fast-bacilli-CSF smears, CSF culture or CSF-PCR assay for *M. tuberculosis*. The T-SPOT. *TB* test (Oxford Immunotec) for tuberculous meningitis diagnosis has a higher sensitivity (62%–100%) than conventional diagnosis techniques and better than that described for TST.[1,4–7] Some authors suggest that IGRAS could be performed in CSF instead of in plasma with better sensitivity.[2,5,6]

In young children, deficiencies in dendritic cell and TH-1-type T-cells function, which play a central role in avoiding lymphohematogenous spread of *M. tuberculosis* after the infection, contribute to the susceptibility to develop disseminated disease.[8] Up to 50% of children with TB meningitis have negative TST results[9] and this is believed to be consequence of diminished functioning of T lymphocytes with less cytokine production. For this reason, in young children with TB meningitis and negative TST we would expect to find a high percentage of indeterminate IGRA results due to absence of response in mitogen control. Nevertheless, our patient presented an initial negative result with a good IFN-$\gamma$ production in the mitogen control, and a positive result after 6 weeks of evolution. Considering that tuberculous meningitis are early developed after the infection,[9] some negative TST and IGRAS results observed will be probably due to prompt spread of the mycobacteria in new acquired infections without an effective T lymphocytes response development. In a child with suspected tuberculous meningitis and a negative IGRA, we believe it is prudent to initiate antituberculous therapy.

## ACKNOWLEDGMENTS

*The authors thank Spanish Pediatric Association (AEP) for the 19th Ausonia-Arbora Grant that has contributed to perform all the studies of the Spanish Collaborative Group for the study of QuantiFERON-TB Gold Test In Tube in children.*

**Ana Méndez Echevarría, MD, PhD**
**Fernando Baquero-Artigao, MD**
Paediatric Infectious Disease Unit
La Paz Hospital

**Miguel González-Muñoz, MD, PhD**
Immunology Department
Carlos III Hospital

**Fernando De Castillo, MD, PhD**
Paediatric Infectious Disease Unit
La Paz Hospital

**Maria José Mellado Peña, MD, PhD**
Paediatric Department
Carlos III Hospital

**Ramón Velázquez-Fragua, MD**
Paediatric Neurology Department
La Paz Hospital
Madrid, Spain

## REFERENCES

1. Liao CH, Chou CH, Lai CC, et al. Diagnostic performance of an enzyme-linked immunospot assay for interferon-gamma in extrapulmonary tuberculosis varies between different sites of disease. *J Infect*. 2009;59:402–408.
2. Luca MC, Petrovici CM, Vata A, et al. Gamma interferon testing in blood and cerebrospinal fluid–rapid method for the diagnosis of tuberculous meningitis [in Romanian]. *Rev Med Chir Soc Med Nat Iasi*. 2008;112:108–110.
3. Iguchi M, Maruyama K, Tsutsumi Y, et al. QuantiFERON: an early diagnostic tool for cerebral tuberculosis [in Japanese]. *Rinsho Shinkeigaku*. 2008;48:259–262.
4. Quan C, Lu CZ, Qiao J, et al. Comparative evaluation of early diagnosis of tuberculous meningitis by different assays. *J Clin Microbiol*. 2006;44:3160–3166.

5. Kosters K, Nau R, Bossink A, et al. Rapid diagnosis of CNS tuberculosis by a T-cell interferon-gamma release assay on cerebrospinal fluid mononuclear cells. *Infection*. 2008;36:597–600.
6. Thomas MM, Hinks TS, Raghuraman S, et al. Rapid diagnosis of *Mycobacterium tuberculosis* meningitis by enumeration of cerebrospinal fluid antigen-specific T-cells. *Int J Tuberc Lung Dis*. 2008;12:651–657.
7. Kim SH, Chu K, Choi SJ, et al. Diagnosis of central nervous system tuberculosis by T-cell-based assays on peripheral blood and cerebrospinal fluid mononuclear cells. *Clin Vaccine Immunol*. 2008;15:1356–1362.
8. Lewinsohn DA, Gennaro ML, Scholvinck L, et al. Tuberculosis immunology in children: diagnostic and therapeutic challenges and opportunities. *Int J Tuberc Lung Dis*. 2004;8:658–674.
9. Carrol ED, Clark JE, Cant AJ. Non-pulmonary tuberculosis. *Paediatr Respir Rev*. 2001;2:113–119.

## ERRATUM

Multiple Brain Abscesses Caused by *Pseudomonas luteola*: ERRATUM

In the Letter to the Editor that appeared on page 1144 of volume 27, number 12, the authors were listed in the incorrect order. The author list should have appeared as Anne Gaschet, PD, Charlotte Engrand, MD, Caroline Piau, PS, Jérémie Violette, PS, Pierre Bétrémieux, PhD, Pierre Tattevin, PhD, Patrick Pladys, PhD, Pierre-Yves Donnio, PhD, and Anne Jolivet-Gougeon, PhD.

Reference:
Gaschet A, Piau C, Violette J, et al. Multiple Brain Abscesses Caused by *Pseudomonas luteola*. *Pediatr Infect Dis J.* 2008;27:1144–1146.