nationally agreed upon. This case illustrates another potential pitfall in melioidosis serodiagnosis. Most isolates express a conserved LPS antigen, which allows use of a single reference strain for determination of anti-LPS antibodies (8). However, because some strains express different LPS antigens, serologic tests must be performed with the patient’s own strain.

This case illustrates potential pitfalls in diagnosing melioidosis, which requires clinical and laboratory awareness and knowledge of its geographic distribution. LPS-based serologic assays should use a range of isolates representative of known LPS types.

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A. J. Brent and R. Handy had clinical responsibility for the patient. R. Handy and P.C. Matthews made the initial microbiologic diagnosis of melioidosis, and T.L. Pitt confirmed the isolate as 

Burkholderia pseudomallei. T.L. Pitt performed the serology and SDS-PAGE analysis of lipopolysaccharide antigens. All authors contributed to preparation of the manuscript. A.J. Brent is guarantor for the article, had full access to all the clinical and microbiologic data, and had final responsibility for the decision to submit for publication.

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Subclinical Plasmodium falciparum Infection and HIV-1 Viral Load

To the Editor: Studies indicate that Plasmodium falciparum infection increases HIV replication in adults (1,2). Although malaria-related illness and death are more common in children, and HIV-1 generally progresses faster in children than in adults (3,4), to our knowledge the effect of intermittent malaria on HIV-1 viral load has not been directly explored in children. To investigate this issue, we monitored HIV-positive infants from 1996–2001 birth cohort study in Kisumu, Kenya, a P. falciparum–holoendemic area.

Study design and methods have been described elsewhere (5,6). Twenty-four children that were perinatally infected with HIV were included in this substudy. During monthly visits during the child’s first 2 years of life, malaria and HIV incidence were recorded (5,6). Both children with malaria-positive blood smears and those with fever but no smear result available were treated with sulfadoxine-pyrimethamine according to national guidelines. At the time of this substudy, none of the study participants were taking antiretroviral drugs.

HIV and malaria diagnoses were determined by using standard methods (5–7). To reduce the chance of including infants infected through breast-feeding, perinatal infection was defined as ≥2 consecutive HIV-positive tests, with the first positive PCR result by 4 months of age (7). The so-called baseline viral load was the premalaria value measured 1 month before the first observation in the analysis. To be included in the analysis, follow-up visits had to have data available on the current and previous months’ viral load and malaria status and occur at roughly monthly intervals at ≥4 months of age. Malaria parasites were found at 53 of 146 visits in the month before viral load measurement, although at 89% of visits in which children were malaria-positive, the children’s samples had <1,000 parasites/µL, and in only 13% of visits in which children had parasitemia they also have fever (8). Median number of observations per child was 7 (range 2–18). No significant demographic or clinical differences were found between HIV-positive children in this substudy and those enrolled in the full cohort (data not shown).

Clinical and demographic variables were evaluated in univariate repeated measures analysis to determine associations with log-transformed HIV-1 viral load. Age and
baseline viral load were strong predictors of current load (Table). Although not statistically significant, clearing the previous month’s malaria infection was associated with a drop in viral load (Table, p = 0.09). It was not possible to distinguish between the effects of treatment versus malaria clearance because 87% of malaria infections were treated with antimalarial drugs. However, viral load increased in those incorrectly treated for malaria presumptively (Table).

After adjusting for age and baseline viral load, we assessed log_{10} HIV viral load in relation to malaria clearance, persistence, absence, or new infection using a repeated measures model with autoregressive covariance structure. No differences were statistically significant, although clearing an infection versus no malaria had a 0.22 log viral load decrease (Table, p = 0.10). When 15 malaria episodes with malaria-free visits 1 month before and after the episode were compared, mean difference (signed-rank test) in viral load “before” and “after” malaria was not significant.

Our findings suggest that low-density malaria infection may not dramatically affect plasma HIV-1 levels in infants. This finding is similar to results of studies of perinatally HIV-infected children in which, although viral loads were unavailable, number of malaria episodes did not significantly affect development of AIDS-related symptoms (9,10). While clinical malaria leads to at least short-term HIV viral load increases in adults (1,2), the effect of subclinical malaria is unclear, and even less is known about coinfection in children. Children usually have higher baseline viral loads than adults; thus, the relative effect of malaria on viral load may not be as great. To reduce the impact of passively transferred maternal antibodies, analyses were done on visits after the child was 4 months old. However, lack of fully acquired antimalarial immunity may have led to different HIV/malaria interactions than seen in adults.

Viral load increased in infants that were incorrectly treated presumptively (due to fever) for malaria (Table). Most of these children were found to have other infections. Fever in malaria-endemic areas is often assumed to be malaria-related, but delay in treatment of nonmalarial infections may be harmful in HIV-infected children. Our assessment was limited in size and duration. Furthermore, in attempting to provide optimal patient care through conducting monthly surveillance and encouraging mothers to bring children in during febrile episodes, ability to assess the effect of high-density malaria was diminished because parasitemia levels never reached clinically significant levels. Finally, because malaria was diagnosed by microscopy, rather than PCR, some subclinical malaria infections may have been misclassified as malaria-negative.

Although we found no evidence of an association between subclinical, 

| Factor                                      | No.† | Predicted beta (SE) | p value |
|---------------------------------------------|------|---------------------|---------|
| Baseline viral load                         |      |                     |         |
| Log viral load per mL                       | 146  | 0.65 (0.09)         | <0.01   |
| Documented fever                            | 23   | -0.02 (0.11)        | 0.85    |
| Temperature ≥37°C                            | 18   | 0.07 (0.14)         | 0.62    |
| Previous visit documented fever             |      |                     |         |
| Temperature ≥37°C                            | 18   | 0.07 (0.14)         | 0.62    |
| Vaccination within 2 weeks of visit         |      |                     |         |
| Yes                                         | 15   | 0.14 (0.14)         | 0.34    |
| Vaccination within 2 weeks of previous visit|      |                     |         |
| Yes                                         | 18   | 0.09 (0.12)         | 0.46    |
| Pneumonia                                   |      |                     |         |
| Present                                     | 22   | 0.01 (0.13)         | 0.93    |
| Anemia                                      |      |                     |         |
| Hemoglobin <8 g/dL                          | 25   | -0.25 (0.16)        | 0.15    |
| Anemia during previous visit                 | 27   | 0.01 (0.13)         | 0.95    |
| Hemoglobin <8 g/dL                          |      |                     |         |
| Antimalarial drugs received at or since previous visit | 59   | 0.02 (0.10)         | 0.84    |
| Malaria status current visit                 |      |                     |         |
| Parasitemia positive                        | 39   | -0.07 (0.10)        | 0.05    |
| Malaria status previous visit               |      |                     |         |
| Parasitemia positive                        | 53   | -0.14 (0.09)        | 0.14    |
| Malaria status and antimalarial drug received at or since previous visit |      |                     | <0.01   |
| Treated parasitemia                          | 46   | -0.11 (0.09)        | 0.25    |
| Did not treat parasitemia                    | 7    | -0.002 (0.17)       | 0.99    |
| Treated, no parasitemia                     | 13   | 0.28 (0.10)         | <0.01   |
| Did not treat, no parasitemia               | 80   | Reference           |         |
| Malaria dynamics (previous vs. current visit)|      |                     | 0.36    |
| Cleared infection‡ (+→)                     | 29   | -0.20 (0.11)        | 0.09    |
| Reinfection/persistence (+ +)               | 24   | -0.16 (0.15)        | 0.28    |
| New infection (−→)                          | 15   | -0.18 (0.14)        | 0.20    |
| No malaria‡ (→→)                            | 78   | Reference           |         |
| Age, mo                                     |      |                     |         |
| 4–9                                         | 73   | -0.39 (0.13)        | <0.01   |
| 10–24                                       | 73   | Reference           |         |

*Statistically significant associations (p<0.05) are indicated in boldface.†Total sample size was 146 follow-up visits. Number of visits with the indicated characteristic are listed. Sample size was 145 for pneumonia and anemia and 144 for anemia during previous visit.‡After multiple comparisons (Tukey-Kramer) between groups in a model of the effect of malaria on log HIV viral load adjusted for age and baseline viral load, no statistically significant differences were found, although clearing an infection versus no malaria had a p value of 0.10.
low-density malaria and infant HIV-1 viral load, the consequences of high-density or clinical malaria need to be explored. If clinical malaria in infants increases HIV-1 viral load as it does in adults (1,2), our study underscores dual benefits of malaria treatment in the context of HIV: 1) keeping malaria in check, and 2) preventing an increase in HIV viral load. Ethical issues prevent prospective studies to assess the impact of coinfection early in life, but alternatives include using animal models or stored specimens.

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African Tickbite Fever in Travelers, Swaziland

To the Editor: African tickbite fever (ATBF), which is caused by Rickettsia africae, is well documented in travelers to southern Africa (1–3) and transmitted by ungulate ticks of the genus Amblyomma. Positive serologic results were reported in 9% of patients (1) and 11% of travelers (4) from southern Africa. We report an outbreak of ATBF with an attack rate of 100% among 12 Dutch travelers to Swaziland.

The 12 travelers (9 male and 3 female) visited Mkhaya Game Reserve in Swaziland in May 2003 for several days. Upon returning to the Netherlands, they consulted our clinic for assessment for fever, malaise, and skin eruptions. Epidemiologic and clinical data were obtained after the patients provided informed consent. All symptomatic patients were treated before serum samples were collected.

Acute-phase and convalescence-phase serum samples were obtained from 8 patients at 3 and 9 weeks, respectively, after symptoms were reported. Only convalescent-phase serum samples were obtained from the other 4 patients. Serologic assays were conducted for screening and confirmation in Rotterdam, the Netherlands (Department of Virology, Erasmus University Hospital) and Marseille, France (Unité des Rickettsies, Faculté de Médecine, Université de la Méditerranée), respectively.

In Rotterdam, immunofluorescence assays for immunoglobulin G (IgG) and IgM against R. conorii, R. typhi, and R. rickettsii were performed with multilow slides on which antigens were fixed (Panbio Inc., Columbia, MD, USA). Serum samples with fluorescent rickettsiae at dilutions ≥1:32 were considered positive.