

## Concurrent *Plasmodium vivax* Malaria and Dengue

**To the Editor:** The first report of a patient with concurrent malaria (*Plasmodium falciparum*) and dengue was recently published in this journal (1). Herein is presumably the first report of concurrent dengue and malaria due to *P. vivax*.

A 27-year-old woman experienced the onset of myalgia on December 11, 2003, 1 day before returning home to California from India after a 3-month sojourn in that country. The following day she had chills and a low-grade fever, and she visited an urgent care center. A presumptive diagnosis of influenza was made, and she was discharged with antipyretic therapy. A single malaria smear was subsequently reported to be negative for *Plasmodium*.

On December 15, she sought treatment at a hospital emergency department at 3:30 A.M. with an oral temperature of 39.5°C. Her leukocyte count was 4,300 × 10<sup>9</sup>/L, hemoglobin level 119 g/L, and platelet count 157,000 × 10<sup>9</sup>/L. A diagnosis of probable viral syndrome was made, and she was discharged with antipyretic therapy. She returned to the urgent care center the following day with a temperature of 38.6°C, and a 10-day course of amoxicillin was prescribed on discharge.

On December 18, she sought treatment from an infectious disease specialist. She had an oral temperature of 39.3°C and was dehydrated, which led to her admission to the hospital. Results of the examination were otherwise unremarkable.

She reported that she had lived in the United States for the last 4 years, after moving there from India. During her recent trip to India, she had spent most of the time in Surat, followed by 3 days in Mumbai. She indicated that she had had malaria several times

while living in India. She received no vaccinations before her trip and took no malaria prophylaxis; she believed she was likely immune and, in addition, she was concerned about taking medications while breastfeeding her 6-month-old child. The child received no prophylaxis or other medical preparation for the trip but remained well.

Her leukocyte count was 4,500 × 10<sup>9</sup>/L with 50% polymorphonuclear leukocytes, 18% band forms, 3% myelocytes, and 1% metamyelocytes. Hemoglobin level was 11.1 g/L, and platelet count was now 98.0 × 10<sup>9</sup>/L. *P. vivax* was seen on blood smear, and the patient was treated with chloroquine with rapid resolution of her fever, followed by administration of primaquine, during which course she avoided breastfeeding. In addition, enzyme immunoassays for dengue virus were performed on December 19 (immunoglobulin G [IgG] 6.55; IgM 4.17) and subsequently repeated on December 31 (IgG 7.29; IgM 1.07), indicating an acute infection. Viral isolation was not attempted.

I agree with Charrel and colleagues (1) that, although only 2 cases have now been reported, concurrent dengue and malaria is probably not a rare event. This conclusion is supported by a recent report from Pakistan (2).

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## Viruses from Nonhuman Primates

**To the Editor:** I read with interest the article by Jones-Engel et al. (1), which described the frequency of viruses infecting temple rhesus macaques. The investigation included the polyomavirus simian virus 40 (SV40), a pathogen recognized to have infected millions of humans who were vaccinated with polio vaccines produced in cultures of rhesus monkey kidney cells (2,3). The authors indicated that technologic advances have improved the specificity of detecting SV40 antibodies and used an enzyme immunoassay based on viruslike particles (VLPs) to perform the analysis (1). However, the specificity of the SV40 enzyme immunoassay is problematic because studies with serum samples from macaques have found that antibodies are cross-reactive with polyomaviruses JCV and BKV (4). In addition, in monkey sera SV40 VLPs correlated with BKV antibodies. Similar conflicting results have been found in human studies that used polyomavirus VLPs assays (3). These limitations are the result of polyomavirus VLPs assays using expression of the VP1 capsid protein (4), a highly homologous gene among JCV, BKV, and SV40 (3). In contrast, modern molecular biology assays are the preferred method for the analysis of SV40 infections (2,3). In addition, these sensitive and specific techniques can provide insights into the distribution of SV40 strains and variants (2,3). This is important because recent data suggest that the biological properties of SV40 strains vary in vivo (5).

Because current evidence shows that SV40 infections are identified in some humans and that the virus is associated with selected human malignancies (2,3), prospective longitudinal studies that use molecular

techniques are needed to examine the prevalence and ecology of SV40. The Institute of Medicine recognizes that the biologic evidence indicates that infections with this DNA virus could lead to cancer in humans and recommends targeted biologic research of SV40 in human populations (2).

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**In response:** Dr. Vilchez (1) raised questions regarding the specificity of the SV40 viruslike particle (VLP)-based ELISA used to detect SV40 infection in temple monkeys (2). Although it is true that SV40 infection can elicit low-level, cross-reactive antibodies that recognize polyomavirus BKV and to a lesser extent polyomavirus JCV VLPs and, conversely, BKV and JCV infection may elicit low-titer SV40 cross-reactive antibodies (3), these antibodies do not pose a problem for serologic diagnosis of infection in natural hosts of these polyomaviruses. Unless one were to hypothesize that BKV and JCV could infect macaques, SV40 VLP-reactive antibodies could not possibly be produced by anything other than an SV40 infection. In addition, SV40 seroreactivity in macaques is generally very strong but cross-reactive responses are weak (3).

Specificity of SV40 seroreactivity in macaques has recently been demonstrated by competitive inhibition assays (4). SV40 reactivity was blocked by incubation of sera with SV40 VLPs but not significantly reduced by incubation with BKV or JCV VLPs. Specificity of BKV and JCV VLP reactivity in human sera has also been demonstrated by preabsorption with VLPs (5) and competitive inhibition assays (4). Thus, BKV VLP seroreactivity can be completely inhibited by BKV VLPs but not by JCV VLPs and vice versa. When polyomavirus VLP ELISAs are used to diagnosis cross-species infection, such as SV40 infection in humans, competitive inhibition assays are necessary to verify specificity of the response. Engels et al. (6), using SV40 VLP serology and competitive inhibition assays, recently reported evidence for possible infection of zoo workers with SV40. We agree with Dr. Vilchez that molecular biology assays, such as PCR, play a valuable role in viral diagnosis. However, these assays also have limitations in terms

of sensitivity and specificity and therefore are best combined with the full range of viral diagnostic techniques to confirm infection. Serologic testing is more suitable than other assays for estimating cumulative infection.

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