contacts with clandestine gold pan-
ners, mainly Brazilian illegal resi-
dents. This population, in which malaria incidence is almost impos-
sible to evaluate, comes from Amapa 
State, where the incidence of malaria is increasing (5). In 2003, 60.9% of 
patients with malaria cases at 
Cayenne Hospital had a Brazilian 
name compared with 35.4% in 2000 
(6). Also, the gold panners diverted 
the river and built basins where vec-
tors could easily multiply (7).

Initial malaria attacks were treated 
with chloroquine or quinine. Five 
patients experienced ≥1 relapses 
(maximum 3 relapses). The relapses 
treated with 50-mg daily doses 
of primaquine for 4 patients and by 
chloroquine for the fifth patient. Two 
patients had relapses after receiving 
primaquine. Primaquine resistance 
information was not available. How-
ever, resistance to primaquine has emerged in P. vivax strains (8).

We recommended that pre-impreg-
nated battlefield uniforms be available 
for French policemen and chemop-
ophylaxis adherence be reinforced by 
directly observed intake by superviso-
ry staff. Relapses of P. vivax malaria 
are a major therapeutic problem, par-
icularly after primaquine therapy.

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Plasmodium vivax Malaria Relapses 
after Primaquine Prophylaxis

To the Editor: Standard treatment 
of patients with Plasmodium vivax 
malaria includes chloroquine, fol-
lowed by primaquine terminal prophylaxis. Reports of true primaquine 
failure and subsequent P. vivax 
relapse are unusual; most suspected 
cases can be ascribed to poor patient 
adherence, recrudescence of a chloro-
quine-resistant strain, or P. vivax rein-
flection. We report a case of P. vivax 
malaria relapse after therapy with qui-
nine, doxycycline, and primaquine, 
and again after treatment with chloro-
quine and primaquine. P. vivax 
relapses after primaquine treatment 
are exceedingly rare in travelers to South America and are a serious ther-
aputic challenge. Our patient was 
subsequently treated with weekly, sin-
gle-dose chloroquine without recur-
rence of symptoms.

A 77-year-old man had fever and 
chills 2 weeks after returning from 
Brazil. These symptoms were accom-
panied by sweating, fatigue, and a 
mild, productive cough. Review of 
systems was notable for dark, con-
centrated urine and a 10-lb weight 
loss. The patient’s 25-day journey 
included Salvador, Manaús, and a 2-
day stay in the Amazon River basin. 
He did not take malaria prophylaxis 
during his trip.

On physical examination, the 
patient was afebrile with blood pres-
sure of 90/53 mm Hg. Cardio-
vascular, pulmonary, and abdominal 
examination results were unremark-
able. Several petechiae were noted on 
both lower extremities. Laboratory 
tests showed the following: leukocyte 
count 6,300 cells/µL, hemoglobin 
level 13.7 g/dL, platelet count 40,000 
cells/µL, serum creatinine level 1.2 
mg/dL, serum alanine aminotrans-
ferase level 63 IU/L, and serum

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Letters reporting cases, outbreaks, or 
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ces. They may have 1 figure or 
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material not previously published and 
include a word count.

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aspartate aminotransferase level 56 IU/L. Thick and thin peripheral blood smears revealed *P. vivax* with a parasitemia level of 0.67%. Although the existence of chloroquine-resistant *P. vivax* in Brazil is debatable, the patient was conservatively treated with quinine, 650 mg, 3×/day and doxycycline, 100 mg, 2×/day for 7 days, followed by primaquine terminal prophylaxis, 30 mg/day for 30 days with complete resolution of symptoms.

In the absence of travel abroad, the patient experienced similar symptoms 5 months later. On the basis of thick and thin peripheral blood smear examination, a relapse of *P. vivax* malaria was diagnosed. He was given chloroquine, 2.5 g over 3 days, followed by primaquine, 30 mg/day for 30 days. Again, the patient’s symptoms resolved.

Four months after treatment (9 months after the initial episode), the patient experienced the abrupt onset of fever, chills, and dark urine. He had a leukocyte count of 5,900 cells/µL, a hemoglobin level of 14.0 g/dL, and a platelet count of 117,000 cells/µL. Repeat thick and thin blood smears showed *P. vivax* with a parasitemia level of 0.993%. Therapy with chloroquine, 2.5 g over 3 days, followed by primaquine termi- nal prophylaxis, 30 mg/day for 30 days with complete resolution of symptoms.

Despite standard dose administration, 1 study suggested substantial intereth- nic differences in peak plasma concentra- tions of primaquine and its major metabolite, carboxyprimaquine (5). Finally, confounding factors such as drug dosing and patient compliance have complicated most failure reports.

Our patient initially received quin- ine and doxycycline, which excluded a chloroquine-resistant infection. In addition, he completed a primaquine regimen of 10.8 mg/kg, which is twice the current recommended dose. In the absence of reexposure, the patient had a relapse 5 months later. His condition was treated with chloroquine and again with high-dose primaquine. He reported strict adherence to the treat- ment regimen, citing the fastidious use of a weekly pill box as evidence. Despite these measures, another relapse occurred 4 months later. This patient’s course suggests *P. vivax* primaquine failure and possible resistance. When high-dose regimens of primaquine (total 5–6 mg/kg) fail, suppressive doses of chloroquine, 300 mg/week for several months to years may be considered. Our patient received chloroquine therapy, 300 mg/week for the past 4 months without evidence of recurrence.

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