a successful hospital course, only 1 with a negative UAT result died. The remaining 29 patients, including 26 with negative UAT results, were subjected to treatment changes and received treatment with non-β-lactam antibiotics. All of these patients’ conditions improved and clinically stabilized, and the patients were discharged from the hospital.

Because 149 (98%) of the 152 patients were alive and discharged from the hospital on day 30 of hospitalization, β-lactam monotherapy appears to be a safe initial treatment of CAP. Although the UAT is not needed to target β-lactam monotherapy, it still appears to be useful in the management of CAP. A positive test result can support treatment with narrow-spectrum β-lactam antibiotics and can be used to prevent unnecessary antibiotic changes. The present study shows that antibiotic changes will most often not be needed in patients with positive test results but will be needed more frequently in patients with negative test results. When a UAT result is negative, diagnostic tests for conventional and atypical pathogens are useful to gain support for the ongoing treatment or suggestions for treatment alterations (table 1).

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Thrombocytopenia and Plasmodium vivax Malaria

Sir—We were amazed by the letter of Rodriguez-Morales et al. [1] on the occurrence of thrombocytopenia in Plasmodium vivax malaria because some potentially misleading messages are reported. First of all, the authors affirm that “thrombocytopenia is infrequently reported for P. vivax malaria” [1, p. 130]. However, in one of the major textbooks on tropical medicine, White [2] reports that “thrombocytopenia is common to all four human malaria” (p. 1235). Likewise, 2 recent studies [3, 4] clearly demonstrate the high predictive value of thrombocytopenia in the diagnosis of imported malaria, with ORs of 12.4 and 16.5, respectively. However, it might be observed that in both of these studies the frequency of Plasmodium falciparum malaria was high (66% in [3] and 90% in [4]) but the high likelihood of thrombocytopenia with P. vivax malaria was shown by Erhart et al. [5] in a study conducted in Thailand with 646 case patients (OR, 12.5). In our recent experience with 117 cases of imported malaria in our ward, observed during a 7-year period (1997–2004), 24 patients (20.5%) had been infected with P. vivax malaria, and, at admission, the median platelet count was 65,000 platelets/μL (authors’ unpublished data). As shown in table 1, in 5 of 6 studies on P. vivax malaria, the median platelet count was consistently <150,000 platelets/μL [6–10]. Therefore, we believe that thrombocytopenia should be considered a common hematological feature of P. vivax malaria.

Furthermorf, we did not observe any case involving bleeding, despite the high percentage of patients affected by thrombocytopenia—an observation that is in keeping with the experiences of Newton et al. [10] and Oh et al. [8]. Because of this, it was unexpected that Rodriguez-Morales et al. [1] reported a high percentage of patients (44% of those with <60,000 platelets/μL) affected by P. vivax malaria who underwent platelet transfusion. They fail to mention the reasons for platelet transfusion; however, it should be noted that after starting treatment for malaria, the platelet count generally rises rapidly; bleeding in the absence of comitant disseminated intravascular coagulation is rare, and platelet transfusion is rarely, if ever, required.

Finally, the rate of anemia at admission reported by Rodriguez-Morales et al. [1] (observed in 96. 6% of patients)—although similar to the rate reported by Kotwal et al. [6] in US rangers—is unusually high. In the majority of reports in the literature, as well as in our experience, anemia occurs in no more than 30% of patients. The high rate of anemia reported by Rodriguez-Morales [1] might be correlated with patients’ delayed presentation, or with the comitant presence of other possible causes (i.e., malnutrition or parasitic diseases). However, it should be emphasized that the association of anemia


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and malaria in the physician’s mind could represent a possible pitfall in the diagnosis of imported malaria in travelers.

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References


Reply to Antinori et al.

We appreciate the comments made by Antinori et al. [1] regarding our letter [2]. The main contribution of our report lies in the occurrence of severe thrombocytopenia attributed to Plasmodium vivax infection, not of thrombocytopenia per se, as Antinori et al. [1] imply. Furthermore, Antinori et al. [1] refer to a textbook to suggest that thrombocytopenia is a common phenomenon found in all four human malarials [3]. Interestingly, the reference used by White [3] as a basis for this particular statement is a publication on thrombocytopenia due to Plasmodium falciparum [4]. Antinori et al. [1] further base their letter on a report of thrombocytopenia due to P. vivax from Thailand [5] that was also referenced in our article [2]. The findings of this study [5] do not discuss cases of severe thrombocytopenia.

We are certain that severe thrombocytopenia in P. vivax infection has rarely been reported. Furthermore, Antinori et al. [1] elaborate most of their conclusions on the basis of a few cases of imported P. vivax malaria (3–4 per year) in a non-immune population. In Sucre, Venezuela, malaria is caused almost exclusively by P. vivax. This region of Venezuela is also considered a zone with moderate-to-high levels of malaria transmission. Therefore, most of the population has suffered previous episodes of P. vivax infection. We believe that making the distinction between immune and nonimmune populations exposed to P. vivax is relevant to the occurrence of severe thrombocytopenia.

Table 1. Summary of data on thrombocytopenia and anemia from series of cases of Plasmodium vivax malaria.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Study location</th>
<th>No. of study patients</th>
<th>Types of study patients</th>
<th>Platelet count data</th>
<th>Anemia, proportion (%) of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>[5]</td>
<td>Thailand</td>
<td>646</td>
<td>Migrant workers with imported cases</td>
<td>Median count (range), platelets/μL: 131,000 (males); 141,000 (females); Proportion (%) of patients with count &lt;150,000 platelets/μL: 86/101 (85); Proportion (%) of patients with count &lt;60,000 platelets/μL: 30/101 (30)</td>
<td>Anemia: 81/646 (12.5)</td>
</tr>
<tr>
<td>[6]</td>
<td>USA</td>
<td>38</td>
<td>US Rangers with imported cases</td>
<td>109,000 (40,000–308,000); 26/31 (90); NR</td>
<td>NR</td>
</tr>
<tr>
<td>[7]</td>
<td>Australia</td>
<td>63</td>
<td>Australian travelers, expatriates, and immigrants with imported cases</td>
<td>109,000; NR</td>
<td>NR</td>
</tr>
<tr>
<td>[8]</td>
<td>South Korea</td>
<td>101</td>
<td>Veterans, travelers, and residents in fected in areas of endemicity</td>
<td>NR; 86/101 (85); 30/101 (30)</td>
<td>NR; 52/101 (51.5)</td>
</tr>
<tr>
<td>[9]</td>
<td>Colombia</td>
<td>104</td>
<td>Rural residents</td>
<td>269,000; 8/104 (8); NR</td>
<td>NR; 44/104 (42.3)</td>
</tr>
<tr>
<td>[10]</td>
<td>USA</td>
<td>97</td>
<td>US Marines with imported cases</td>
<td>109,000; (83)*; NR</td>
<td>NR</td>
</tr>
<tr>
<td>Present</td>
<td>Italy</td>
<td>24</td>
<td>Italian nationals who traveled abroad with imported cases</td>
<td>65,000 (28,000–352,000); 23/24 (96); 11/24 (45.8)</td>
<td>7/24 (29.1)</td>
</tr>
</tbody>
</table>

NOTE. NR, not reported.

* Includes 7 patients with cases of P. falciparum malaria.

† Anemia was defined as a hemoglobin level of <12 g/dL, except in [6], in which it was defined as a hematocrit of <42 %, and in [5], in which it was defined as an RBC count of <4,000,000 cells/μL.

* Authors’ unpublished data.