Travel-acquired infections in general

To the Editor—I am deeply impressed by the articles that appeared in the Hong Kong Medical Journal and other journals on the subject of severe acute respiratory syndrome (SARS). Our front-line doctors have proven their professionalism in every respect and deserve our utmost admiration and gratitude. Now that the epidemic has subsided, at least temporarily, it might be a time to reflect on the problem of travel-acquired or ‘imported’ infection on a broad perspective (one might use the term ‘extra-territorially acquired infection’ in contrast to ‘community-acquired infection’). Severe acute respiratory syndrome may return, or another infection—old or new—may be introduced to our community, especially with the intensified integration of Hong Kong into mainland China. Travel-acquired infection may be at least as important as community-acquired infection. In recent years, paragonimiasis and melioidosis have been reported to have been introduced to our community, especially with the intensified integration of Hong Kong into mainland China. Travel-acquired infection may be at least as important as community-acquired infection. In recent years, paragonimiasis and melioidosis have been reported to have been spread by patients who had been travelled to eastern China and Thailand, respectively.5,6

I reviewed the records of my hospital practice and found five cases of travel-acquired fever in the run-up to mid-February 2003, when we recorded our first authentic case of SARS in Hong Kong. In two patients, the cause of fever was obvious. One case was due to a post-insect bite carbuncle acquired in Donguan, and the other was a case of cellulitis from a traffic injury acquired in Shenzhen. The remaining three cases deserve further discussion (Table). All three patients shared some common features. They all presented within 12 hours after the onset of fever, having been driven by the severe chills and myalgia. Their chest X-ray films all showed some bronchitic change, but no florid pneumonitis. In all patients, fever was refractory to a combination of intravenous piperacillin-tazobactam and azithromycin, but responded within 12 hours to a combination of oral ribavirin, methisoprinol, and intramuscular gammaglobulin. By day 4, all patients were afebrile. From their response to treatment, they probably had acquired some form of virus infection during their travels. In particular, one patient had remarked that nearly all fellow travellers in his boat tour of the Three Gorges came down with fever. In May, when virus testing became more available, I traced one of the two patients who had been to the Three Gorges and I tested her for coronavirus antibody; her test result was negative.

Obviously, we need more organised efforts to protect our community from travel-acquired and imported infections in terms of public education, pre-tour advisories, and (when applicable) immunisation, vigilant monitoring of returning travellers, and prompt quarantine and isolation measures whenever indicated. The same measures should also apply to visitors and immigrants. The relevant health authority should act as coordinator between established viral laboratories in the territory and the medical community at large so that sera could be promptly obtained, tests could be readily available and carried out, and the results rapidly reflected to all parties concerned.

Acknowledgement

I am grateful to Dr J Chan, Department of Medicine, University of Hong Kong, for reviewing this letter.

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References


Table. Characteristics of three patients with travel-acquired fever

<table>
<thead>
<tr>
<th>Date presented</th>
<th>Sex/age (years)</th>
<th>Temperature/ duration of fever</th>
<th>White blood cell count (x 10^9/L)/lymphocytes</th>
<th>Site visited</th>
</tr>
</thead>
<tbody>
<tr>
<td>14 November 2002</td>
<td>M/90</td>
<td>37.6°C, 4 days</td>
<td>10.1/21.4%</td>
<td>Zhuha</td>
</tr>
<tr>
<td>9 February 2003</td>
<td>M/66</td>
<td>38.9°C, 36 hours</td>
<td>7.15/9.5%</td>
<td>Three Gorges</td>
</tr>
<tr>
<td>20 February 2003</td>
<td>F/65</td>
<td>37.3°C, 12 hours</td>
<td>4.18/46.0%</td>
<td>Three Gorges</td>
</tr>
</tbody>
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