Imported Malaria Treated in Melbourne, Australia: Epidemiology and Clinical Features in 246 Patients

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Background: Imported malaria is increasing in nonendemic countries, including Australia. The objective of this study was to describe the epidemiology and clinical features of travelers with imported malaria presenting to a specialist infectious diseases hospital.

Methods: A retrospective case series of 246 consecutively admitted inpatients with laboratory confirmed malaria. The main outcome measures were the proportion of patients infected with each malaria species, and relationship between species and country of birth, area of acquisition, adequacy of chemoprophylaxis, clinical features, laboratory investigations, and treatment.

Results: Plasmodium vivax caused 182 (68.9%) episodes, *Plasmodium falciparum* caused 71 (26.9%), *Plasmodium ovale* caused 5 (1.9%), and *Plasmodium malariae* 1 (0.4%). Fifty-six percent of patients reported chemoprophylaxis use. People born in a country with endemic malaria (36.6%) were less likely to have used chemoprophylaxis. Malaria was most commonly acquired in Papua New Guinea and Southeast Asia. The median times to diagnosis after return to Australia for *P. falciparum* and *P. vivax* infections were 1 and 9 weeks respectively. The longest interval between last arrival in Australia and presentation with *P. falciparum* malaria was 32 weeks. Fever (96%), headache (74%), and a tender or palpable spleen (40%), were the most common clinical features. Diarrhea was more common in *P. falciparum,* and rigors in *P. vivax* infections. Thrombocytopenia (71%), abnormal liver function tests and an elevated C-reactive protein (85%) were common. Six patients had severe falciparum malaria but no deaths occurred during the study period.

Conclusions: Malaria remains a health threat for those traveling in endemic areas and is associated with failure to use chemoprophylaxis appropriately. Nonspecific clinical features may lead to delayed diagnosis and misdiagnosis. Malaria should be suspected in the febrile traveler, regardless of birthplace, prophylaxis, symptomatology, or the time that has elapsed since leaving the malarious area.

The rising incidence of imported malaria in developed countries[1,2] reflects increasing travel in tropical areas, where malaria is endemic and is a major cause of morbidity and mortality.[3,4] Each year more than 1.45 million people arrive in Australia from malarious areas[5] and, although Australia was declared free of endemic (indigenous) malaria in 1981,[6] there have been 7,381 cases reported in the last decade (Fig.), with 7 deaths.[7] During the same period in the United States, there were 12,386 cases of malaria with 63 deaths.[8]

Returned travelers, migrants, and overseas visitors with malaria, usually present within days or weeks of arriving in a nonendemic country with the classic features of fever and headache.[9] Atypical clinical features or delayed presentation[10] may make diagnosis difficult and every effort must be made to examine multiple blood smears to exclude the infection. Inadequate or no chemoprophylaxis,[11] as well as delayed diagnosis, may increase

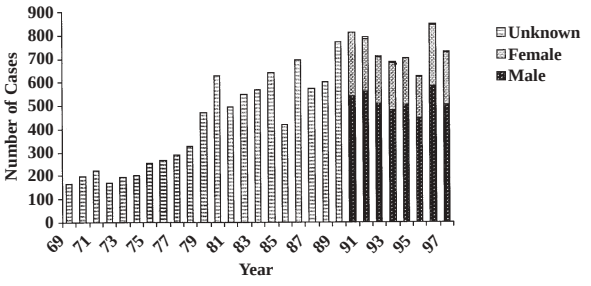

Figure Total number of malaria cases in Australia by year.

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the risk of severe *Plasmodium falciparum* infection and result in life-threatening cerebral malaria, renal failure, coagulopathy or prolonged hypoglycemia.

This study was undertaken to document the epidemiological and clinical features of patients with imported malaria in Melbourne, Victoria. Approximately 60% of the 460 malaria cases reported in the state of Victoria during the study period were admitted to Fairfield Infectious Diseases Hospital, a specialist infectious diseases teaching hospital, providing a unique opportunity for clinical data collection.

Methods

Study Design

This study was designed as a retrospective case series.

Patient Selection

The medical records of all patients admitted to Fairfield Hospital with a clinical diagnosis of malaria (International Statistical Classification of Diseases 9 code of 084.0-084.6) between January 1, 1990 and June 30, 1994 were retrospectively examined by one of two authors (PR or MT). A pro forma was used to record abstracted data relating to demographic details, travel history, chemoprophylaxis, clinical features, laboratory tests and management. All patients for whom there was microscopic confirmation of malaria were included in the study. Staff in the malaria reference laboratory at the Victorian Infectious Diseases Reference Laboratory examined all thick and thin blood films. All identified records were crosschecked with laboratory logbooks to ensure that no cases were missed. Severe falciparum malaria was defined according to WHO criteria[12] (i.e., parasitemia and one or more of unrousable coma, repeated generalized convulsions, pulmonary edema or acute respiratory distress syndrome, glucose $\geq$ 2.2 mmol/L, systolic blood pressure $<$ 70 mm Hg, creatinine $>$ 0.265 μmol/L, arterial pH $<$ 7.25, hemoglobin $<$ 5 g/dL, hemoglobinuria, or spontaneous bleeding. A parasitemia $>$ 5% was also included as a defining criteria as nonimmune patients predominated in the study group[12]). The adequacy of antimalarial chemoprophylaxis was assessed by the authors using guidelines that were current during the study period.[2,13,14]

Data Handling and Analysis

Data relating to social background, travel history, clinical history, diagnostic procedures, treatment and outcome details, were abstracted and entered into Microsoft Excel. Data were subsequently exported to Epi-Info 6.04[15] for descriptive analysis and hypothesis testing. The chi-squared statistic with Yates' correction for continuity, relative risks (with 95% confidence limits), or Fisher exact statistic, were used for categorical data. A 2-sample *t*-test, or analysis of variance were used to compare the means of two or more groups for normally distributed data, and the Kruskal-Wallis H test was applied when the data had a nonnormal distribution.

Results

Two hundred and forty-six people were admitted to Fairfield Hospital with confirmed malaria on 264 occasions. There were 158 (64.2%) males and 88 (35.8%) females. The median age was 29 years. Ninety (36.6%) were born in a country with endemic malaria and 54% of these acquired malaria in India, Sri Lanka, Pakistan, or Southeast Asia. One hundred and thirty-three were Australian-born, of whom 51% acquired malaria in Papua New Guinea (PNG), Bougainville, or the Pacific Islands (Table 1).

Chemoprophylaxis

One hundred and thirty-nine people (56%) took malaria prophylaxis. Of these, only 29% were assessed by the authors (AJ, BB) to have taken the drugs according to manufacturers' recommendations (i.e., took medications regularly during, and for 4 weeks after, travel at the recommended dose). Even if taken as recommended, the antimalarial prophylaxis was assessed by the authors as inadequate protection against *P. falciparum* in 76% of cases because of likely drug resistance in the area visited. Prophylaxis regimens included chloroquine alone (n = 37), chloroquine and Maloprim (pyrimethamine/dapsone, n = 57), Maloprim alone (n = 4), mefloquine (n = 26), and doxycycline (n = 5). People born in malaria-endemic countries were less likely to have used chemoprophylaxis (15/84) compared with those from nonendemic countries (124/147, Relative Risk [RR] 5.25, 95% confidence intervals (CI) 3.56–7.74, $p < .001$).

Five of the 26 people who took mefloquine prophylaxis had *P. falciparum* infection. None of these took prophylaxis for the recommended 4 weeks after leaving the malarious area. (One person who had traveled for a month in Ghana developed infection while still taking mefloquine, and 3 others 11, 14, and 21 days respectively after its cessation). Twenty of the mefloquine users had *Plasmodium vivax* and one had *Plasmodium ovale* infection. Six of these developed symptoms within 4 weeks of returning to Australia, but only one was a true mefloquine failure. This case, a 16-year-old male student who had camped in northwest PNG for 1 month, developed

Table 1 Region or Country of Likely Acquisition of Malaria in 246 Patients Admitted to Fairfield Hospital

Place of Birth	*Australia (%)*	*US, NZ, Europe (%)*	*Countries with Endemic Malaria (%)*	*Total (%)*
Number of People with Malaria	133 (54)	23 (9)	90 (37)	246
Area of Travel				
Southeast Asia*	32 (24)	9 (39)	20 (22)	61 (25)
Asia†‡	5 (4)	2 (9)	29 (32)	36 (15)
Africa§#	20 (15)	3 (13)	9 (10)	32 (13)
PNG, Bougainville	55 (41)	7 (30)	24 (27)	86 (35)
Solomon Islands and Vanuatu	13 (10)		8 (9)	21 (9)
Central, South and North Americas	4 (3)			4 (2)
Multiple countries¶	4 (3)	2 (9)		6 (2)

*Southeast Asia: Indonesia, Myanmar, Thailand, Malaysia, Indochina, or not otherwise specified.

†Asia: refers to travel in India, Sri Lanka, or Pakistan.

‡Includes 5 who had traveled in both southeast Asia and Asia.

§Africa: Ghana, Kenya, Nigeria, Sudan, Tanzania, Zaire, Zambia, Zimbabwe, East Africa, or not otherwise specified.

#Includes 3 people who had also been in Asia and 4 who had been in Indonesia.

¶Multiple countries refers to travel in at least 3 of the other areas in the Table.

P. vivax infection 2 weeks after leaving PNG, while still taking mefloquine. Five people taking doxycycline developed malaria after ceasing the drug: 3 had *P. vivax,* 1 *P. falciparum,* and 1 *P. ovale* infection. None took doxycycline for 4 weeks after leaving the risk area, as recommended by the manufacturer.

Parasite Species and Country of Acquisition

P. vivax was the causative organism in 182 (68.9%) episodes, *P. falciparum* in 71 (26.9%), *P. ovale* in 5 (1.9%), and there was 1 (0.4%) case of *Plasmodium malariae.* Five people had mixed infections: *P. vivax/P. falciparum* in 4 (1.5%) and *P. falciparum/P. ovale* in 1 (0.4%). All 264 episodes of malaria were microscopically confirmed on admission. In 10 episodes, a different species of *Plasmodium* was identified to that diagnosed elsewhere.

The likely countries or regions where malaria was acquired are shown in Table 1. Twenty-seven (25%) of 107 people who traveled in PNG, the Solomon Islands or Vanuatu, and 22 (69%) of 32 who traveled in Africa were diagnosed as having *P. falciparum* malaria. Travel in Africa was the only significant association between geographic area and species ($p < .0001$).

Duration of Illness before Diagnosis

The median times to diagnosis after the most recent return to Australia for *P. falciparum* and *P. vivax* infections were 1 and 9 weeks respectively. *P. falciparum* was more likely to be the diagnosis if the patient had returned to Australia in the last 3 weeks (RR 5.95, 95% CI 3.63–9.75, $p < .0001$). However, 10 (14%) people with *P. falciparum* infection had arrived in Australia more than 4 weeks previously, and 8 (11%) of these had arrived more than 8 weeks before presentation. The longest interval between last arrival in Australia and presentation with malaria for *P. falciparum* was 32 weeks, and for *P. vivax* was more than 2 years. *P. vivax* was significantly more likely if presentation was more than 3 weeks after return (RR 2.31, 95% CI 1.75–3.04, $p < .001$).

The median duration of symptoms prior to the patient presenting for diagnosis was 4 days for *P. falciparum* (range 1–42 days) and 6 days for *P. vivax* (range 1–42 days). If the patient presented for initial consultation (not necessarily at Fairfield Hospital) within 4 days of the start of symptoms, *P. falciparum* infection was significantly more likely to be the diagnosis than *P. vivax,* (RR 1.9, 95% CI 1.26–2.86, $p = .003$).

Treatment before Admission to Fairfield Hospital

Some form of treatment was commenced in 174 episodes prior to admission to Fairfield Hospital. In 42 episodes the diagnosis of a bacterial or viral illness had resulted in treatment with antibiotics (24) or panadol (18), and in 70 episodes antimalarial chemotherapy was commenced. In 62 patients the type of treatment was unknown. The duration of this prior treatment was recorded in 160, of whom 120 (69%) had been treated for 2 days or less. In 15 (9%) episodes, treatment had continued for 7 days or more, prior to presentation at Fairfield Hospital. Only 3 of these 15 had received antimalarial therapy (all had *P. vivax*). Three of the 15 had *P. falciparum* infection and none of these had received antimalarial therapy. They presented with parasitemias of 0.6, 1.8, and 47% respectively.

Clinical Features and Treatment at Fairfield Hospital

The main clinical features of the 264 episodes are shown in Table 2. Overall, fever (temperature ≥ 37.5°C) was documented in 187 (70.8%) cases, and in 76 cases where fever was not recorded 75 individuals reported

Table 2 Symptoms and Physical Signs Recorded for 264 Consecutive Malaria Episodes

Symptom/Sign	*Total (%) n = 264**	P.vivax *(% of Pv episodes) n = 182*	P.falciparum *(% of Pf episodes) n = 71*
Headache	194 (73.5)	137 (75.3)	49 (69)
Rigors	154 (58.3)	117 (64.3)	32† (45.1)
Sweats	101 (38.3)	72 (39.6)	27 (38)
Nausea and/or vomiting	99 (37.5)	64 (35.2)	34 (47.9)
Myalgia	91 (34.5)	68 (37.4)	22 (31)
Diarrhea	47 (17.8)	25 (13.7)	21‡ (29.6)
Arthralgia	46 (17.4)	30 (16.5)	11 (15.5)
Cough	24 (9.1)	16 (8.8)	8 (11.3)
Drowsy	16 (6.1)	7 (4)	8§ (11.3)
Fever on admission ≥ 37.5°C	187 (70.8)	129 (70.9)	55 (77.5)
Fever ≥ 39.5°C on admission	49 (18.6)	34 (18.7)	14 (19.7)
Spleen (palpable or tender)	107 (40.5)	67 (36.8)	22 (31)
Tachycardia (≥ 100/minute)	100 (37.9)	68 (37.4)	27 (38)
Tachypnea (≥ 22/minute)	83 (32.4)	60 (33)	19 (26.8)
Hepatomegaly (palpable)	79 (29.9)	55 (30.2)	18 (25.4)
Hypotension (systolic BP < 95 mm Hg)	16 (6.1)	13 (7.1)	3 (4.2)

*Includes *P. ovale* and *P. malariae* episodes.

No significant differences between species except:

†Rigors less common in *P. falciparum* (RR 0.70, 95% CI 0.53–0.93, p = .008)

‡Diarrhea more common in *P. falciparum* (RR 2.15, 95% CI 1.29–3.59, p = .006)

§Drowsiness more common in *P. falciparum* (RR 2.93, 95% CI 1.1–7.78, p = .04)

rigors and/or sweats before admission. One PNG-born schoolgirl had no fever or history suggestive of fever, but had malaria previously and was likely to have been semi-immune. Rigors were reported in 154 (58.3%) cases and were less common in *P. falciparum* infections compared with *P. vivax* (RR 0.70, 95% CI 0.53–0.93, p = .008). Gastrointestinal symptoms were reported in 137 (52%) cases. Although diarrhea was reported in only 47 (18%) cases it was more common with *P. falciparum* (21/71) than *P. vivax* (25/177) infections (RR 1.90, 95% CI 1.27–2.84, p = .006). This association was still significant after exclusion of 13 patients with coincidental fecal pathogens. There were 6 patients with severe falciparum malaria (8.4% of *P. falciparum* episodes) according to WHO criteria.[12] Several patients had more than one criterion; these included severe anemia (1), pulmonary edema (1), renal failure (2), and hyperparasitemia (4). There were no deaths.

A variety of standard treatment regimens including quinine and Fansidar or doxycycline, chloroquine, and chloroquine and primaquine, were used for these patients, depending on the species of *Plasmodium* and the initial response to therapy.

Laboratory Results

The median parasitemia for *P. falciparum* was 0.33% (range 0.0001–47%), for *P. vivax* was 0.20% (range 0.0001–1.78%), and for *P. ovale* was 0.01% (range 0.01–0.02). The first thick and thin smear at Fairfield Hospital was negative in 5 patients. All had self-medicated or received antimalarial therapy from an outside source. Seven patients with *P. falciparum* had a parasitemia of ≥ 5%. One hundred and fifty-eight patients had an abnormal C-reactive protein (CRP) (≥ 10 mg/L) and in 64 it was ≥ 100 (mean 87.2, range 7–309). One hundred and sixty-nine patients had thrombocytopenia, and severe thrombocytopenia (platelets ≥ 50×10^9/L) was more common in *P. falciparum* infections (RR 2.35, 95% CI 1.09–5.08, p < .05). Two patients had Glucose-6-diphosphate (glucose-6-phosphate dehydrogenase) deficiency. Other hematological and biochemical investigations are shown in Table 3.

Duration of Hospitalization and Readmissions

There were 48 admissions where inpatient management was required for 5 days or longer. Fifteen had *P. vivax* and 33 had *P. falciparum* infection. *P. falciparum* was significantly more likely to cause a hospital stay of more than 4 days when compared with *P. vivax* infection (RR 5.64, 95% CI 3.27–9.73, p < .0001). The gender of the patient, the country of birth (malarious or nonmalarious), the use of prophylaxis, and the duration of symptoms before treatment were not associated with a hospital stay of more than 4 days. Fever persisted for 5 days or more after commencement of antimalarial chemotherapy in 8 patients (range 0–13 days, median 1 day). All but one of

Table 3 Laboratory Abnormalities in 264 Episodes of Malaria

Test	*Number Tested*	*Abnormal (%)*
C-reactive protein (≤ 10 mg/L)*	186	158 (84.9)
Total bilirubin (< 17 μmol/L)	212	160 (75.5)
–conjugated bilirubin (< 8 μmol/L)	160	136 (85)
Platelets (< 150 × 10^9/L)	238	169 (71.0)
Alkaline phosphatase (> 90 U/L)	206	50 (24.3)
Alanine aminotransferase (< 50 U/L)	222	42 (18.9)
Leukopenia (WCC < 4.0 × 10^9/L)	262	49 (18.7)
Hemoglobin (< 11 g/dL)	263	45 (17.1)†
Leukocytosis (WCC > 9.0 × 10^9/L)	262	17 (6.5)
Glucose (< 3.3 mmol/L)	21	0

*Normal values are shown in parentheses.

†6 patients had hemoglobin < 8 g/dL on the day of admission.

these patients had *P. falciparum* infection. Blood cultures were taken for 7 of these patients and were negative.

Fifteen people were readmitted to Fairfield Hospital with recurrent malaria during the study period. Twelve were admitted twice, and 3 were admitted three times. Three of the 15 became reinfected when they returned to a malarious country. One patient with *P. vivax* infection who did not receive primaquine therapy was readmitted after 4 weeks with recurrent *P. vivax*. Six patients (4%) with *P. vivax* infection (from Southeast Asia [3], PNG [2], and the Solomon Islands [1]) who had received chloroquine and primaquine therapy were readmitted with recurrent *P. vivax*. Four patients with *P. falciparum* represented between 7 and 36 weeks after treatment with *P. vivax* infection. Three of these had received primaquine therapy. One patient had quinine-resistant *P. falciparum* malaria acquired in Thailand. He was admitted three times with recurrent parasitemia. On the first two occasions he was treated with quinine and doxycycline and on the third occasion was treated successfully with halofantrine and doxycycline.

Discussion

We describe here the epidemiological and clinical features of 264 consecutive patient admissions with imported malaria, representing the largest Australian case series reported, and over half of the cases of malaria notified in the state of Victoria during the study period.

Papua New Guinea, the Solomon Islands and Vanuatu, were most frequently recorded as the presumed areas in which malaria was acquired in this series (44%). This is likely to be a significant association as the total number of travelers arriving in Australia directly from these countries in 1994/1995 constituted only 11% of travelers returning from malaria-endemic areas during that 12 month period.[5] The high incidence of malaria in Australian residents returning from these countries has been recorded in previous reports. In one of these the risk of malaria in those returning from PNG and the Solomon Islands was estimated as 5–6 times that of residents returning from Africa.[16,17]

Antimalarial chemoprophylaxis prevents and reduces the severity of malaria if appropriate medications are taken for the areas visited, and if the drug(s) are taken regularly during travel and for 4 weeks after leaving the malaria endemic area.[18–20] Several factors resulted in 139 people in this study acquiring malaria despite having used chemoprophylaxis. Chemoprophylaxis does not prevent recurrences of and late-onset *P. vivax* malaria, and 113 people (46%) developed *P. vivax* at least 4 weeks after return to Australia. Importantly, less than one-third who took prophylaxis, and for whom information was available, took the medications regularly during, and for the required period after, travel. Even though more than half of the 139 cases were prescribed the correct drug(s) according to guidelines that were current during the study period (1990–1995),[21] it is now evident that the use of chloroquine and/or Maloprim was insufficient protection against *P. falciparum* malaria in many areas during this period because of rapidly developing parasite resistance to these drugs.[2,14,22] As the study was conducted in the first half of the 1990s, information collected about the types of drugs used for chemoprophylaxis is now less relevant. The current CDC and WHO guidelines have been simplified, as Fansidar and Maloprim are no longer routinely used. Mefloquine or doxycycline is now the usual agent of choice if chemoprophylaxis is required.[18–20]

Settlers and visitors from malaria-endemic areas (37% of our series) were identified as a group significantly less likely to take chemoprophylaxis. Antimalarial prophylaxis would not have been prescribed as a routine in their home country, and so those who had recently arrived would have been susceptible to acute malaria acquired before departure, or to relapses of *P. vivax* and *P. ovale*. In addition, settlers who have had previous exposure to malaria may not perceive the need for chemoprophylaxis for home visits. However, immunity to clinical malaria is short-lived (months rather than years), and malaria in those living abroad and returning home for short visits is well documented.[2,10,23]

P. vivax and *P. falciparum* infection are clinically indistinguishable, although the finding that diarrhea occurred more frequently in *P. falciparum* is of interest and has been reported elsewhere.[10,24] Rigors were more common in *P. vivax* infections, which is in keeping with the known rapid synchronization of *P. vivax* asexual stage parasites. *P. vivax* infection is also more likely to present with classical paroxysmal fever (cold, hot, sweaty stages) than *P. falciparum,* which often presents in a more atypical fashion. We found no association between *P. vivax* infections and high temperature on admission (≥ 39.5°C), although others have reported this finding.[10]

P. falciparum did cause significant morbidity with 46% of patients requiring a hospital stay of 5 days or longer. Six patients met WHO criteria[12,23] for severe falciparum malaria, which is similar to other reports,[10,11] and 3 required exchange transfusion for hyperparasitemia. The selection of patients admitted to an infectious diseases hospital may have led to an identification bias as these cases may have been sicker than those admitted elsewhere. However, the proportion of *P. falciparum* patients in this series was similar to that reported in two series from Queensland and New South Wales.[16,25]

There were no deaths in this study but there were 3 deaths elsewhere in Victoria between 1990 and 1996 due to *P. falciparum,* emphasizing the need for early detection and prompt treatment of imported malaria infections.[9] The finding of thrombocytopenia (71% of episodes in this series) should alert medical practitioners to the possibility of malaria even if the initial thick and thin blood smears are negative.[10,26]

The number of malaria cases imported into developed countries is likely to continue to increase due to increasing numbers of travelers to malaria-endemic areas each year, and the growing problem of parasite resistance to antimalarial drugs. Accurate surveillance of both the epidemiological and clinical features of imported malaria infections must therefore remain a public health priority. Medical practitioners need to be vigilant in excluding the diagnosis of malaria in patients presenting with fever, or a history of chills, sweats or rigors, and who have traveled in a malaria endemic area in recent weeks or months.

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References

1. Muentener P, Schlagenhauf P, Steffen R. Imported malaria (1985–1995): trends and perspectives. Bull World Health Organ 1999; 77:560–566.
2. Behrens RH, Curtis CF. Malaria in travellers: epidemiology and prevention. Br Med Bull 1993; 49:363–381.
3. World Malaria Situation in 1993. Commun Dis Intell 1996; 20:88–92.
4. Olliaro P, Cattani J, Wirth D. Malaria, the submerged disease. JAMA 1996; 275:230–233.
5. Bureau of Statistics. Total movement (overseas arrivals) financial year 1995. Commonwealth of Australia, Melbourne, Australia, 1995.
6. Currie B. The diagnosis and treatment of malaria in Australia. Aust Fam Physician 1993; 22:1773–1779.
7. Communicable Diseases Network, Australia and New Zealand, National Notifiable Diseases Surveillance System, Canberra: Dept. of Health and Aged Care, 1999.
8. Anonymous. Summary of notifiable diseases, United States, 1998. MMWR 1999; 47(53):1–93.
9. Kain KC, Harrington M, Tennyson S, Keystone JS. Imported malaria: prospective analysis of problems in diagnosis and management. Clin Infect Dis 1998; 27:142–149.
10. Svenson JE, MacLean JD, Gyorkos TW, Keystone J. Imported malaria. Clinical presentation and examination of symptomatic travelers. Arch Intern Med 1995; 155:861–868.
11. Wetsteyn JCFM, de Geus A. Falciparum malaria, imported into The Netherlands, 1979-1988. Trop Geogr Med 1995; 47(3):97–102.
12. World Health Organization. Severe and complicated malaria. Trans R Soc Trop Med Hyg 1990; 84(Suppl 2):1–65.
13. Rogerson S, Biggs B-A, Brown G. Chemoprophylaxis and treatment of malaria. Aust Fam Physician 1994; 23:1696–1709.
14. Yung AP, Ruff TA. Manual of travel medicine. Melbourne: Victorian Infectious Diseases Service, Royal Melbourne Hospital, 1999.
15. Dean AG, Dean JA, Coulombier D, et al. Epi-Info 6.02. A word processing database and statistics system for epidemiology on microcomputers. c/o CDC Atlanta, Georgia: USD Inc, Stone Mountain, 1995.
16. Hanna JN, Brookes DL, Ritchie SA, et al. Malaria and its implications for public health in far north Queensland: a prospective study. Aust N Z J Public Health 1998; 22:196–199.
17. Report of the Australian Malaria Register for 1990. Commun Dis Intell 1991; 15:400–409.
18. World Health Organization. International travel and health. Vaccination requirements and health advice. Geneva: WHO, 2000. Available on-line at http://www.who.int/dsa/justpub/justpub.htm#International Travel and Health.
19. Centres for Disease Control and Prevention. Health information for international travel 1999–2000, Atlanta, GA: Department of Health and Human Services, 1999. Available on-line at http://www.cdc.gov/travel/yellowbk99.pdf.
20. CDR Guidelines for the prevention of malaria in travellers from the United Kingdom. CDR Rev 1997; 7:137–152.
21. Hall R. Malaria prevention. Med J Aust 1992; 156:103–108.
22. White NJ. Malaria. In: Cook GC, ed. Manson's tropical diseases. 20th Ed. London: WB Saunders, 1996: 1087–1164.
23. Molyneux M, Fox R. Diagnosis and treatment of malaria in Britain. BMJ 1993; 306:1175–1180.
24. Prasad R, Virk K. Malaria as a cause of diarrhoea—a review. PNG Med J 1993; 36:337–341.
25. Boreham R, Relf WA. Imported malaria in Australia. Med J Aust 1991; 155:754–757.
26. Lynk A, Gold R. Review of 40 children with imported malaria. Pediatr Infect Dis J 1989; 8:745–750.