

## Lesson of the Week

# Absence of fever in non-immune patients developing falciparum malaria

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Most doctors would agree that fever which develops either in Africa or on return from Africa must be regarded as malaria until proved otherwise. In the absence of fever, however, the diagnosis may not be considered. We report three cases in which non-immune people who either visited or worked in Malawi without taking malaria prophylaxis developed a non-febrile illness caused by *Plasmodium falciparum*.

### Case reports

**Case 1**—A 69 year old white woman who had lived in Malawi for several years developed malaise and lethargy. Seven days later after an episode of watery diarrhoea she collapsed at home and was admitted to hospital. On examination she was found to be cold, clammy, and confused. Vital signs were: body temperature 35°C, pulse 100 beats/min, and blood pressure 110/70 mm Hg. An electrocardiogram was normal. She improved initially with parenteral fluids but 12 hours later became comatose and shocked. Vital signs were: temperature 35°C, pulse 140/min, blood pressure 60/40 mm Hg. Other clinical data, full blood count, blood glucose and plasma electrolyte concentrations, serum amylase activity, cerebrospinal fluid, and an electrocardiogram were normal. Platelet count was  $45 \times 10^9/l$  and plasma urea concentration 18.8 mmol/l. A clinical diagnosis of overwhelming septicaemia with disseminated intravascular coagulation was made and treatment begun with parenteral antibiotics, corticosteroids, and plasma expanders. She remained afebrile and over the next 12 hours her vital signs and conscious level improved. A thick blood film then showed numerous asexual forms of *P falciparum* (100/high power field). A diagnosis of algid malaria was made. Treatment was given with parenteral quinine dihydrochloride 600 mg every eight hours followed by a pyrimethamine-sulfadoxine and mefloquine combination. Antibiotics and corticosteroids were stopped because of negative blood cultures. Parasitaemia disappeared 92 hours after the start of treatment but the infection was complicated by acute renal failure necessitating peritoneal dialysis, disseminated intravascular coagulation requiring fresh blood transfusions, and jaundice as a result of hepatic dysfunction. She eventually made a good recovery.

**Case 2**—A 59 year old white man resident in Malawi who had complained of anorexia and malaise for three days collapsed during venesection and was admitted to hospital. On examination he was found to be clammy with a temperature of 35.5°C, pulse 88/min, and blood pressure 75/40 mm Hg. Other clinical data, an electrocardiogram, and cardiac enzyme values were normal. Eight hours later he was slightly confused, temperature was 35.6°C, and blood pressure 75/50 mm Hg. A thick blood film contained numerous asexual forms of *P falciparum* (50-100/high power field). Parasitaemia disappeared after 72 hours with parenteral quinine dihydrochloride followed by pyrimethamine-sulfadoxine. Clinical recovery was delayed by right lobar pneumonia, which developed three days after admission but which responded to antibiotics.

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**Plasmodium falciparum malaria may present as a non-febrile illness in a non-immune person; blood smears should be performed in any patient who becomes ill with or without fever after visiting a tropical country**

**Case 3**—A 42 year old white man who was visiting Malawi developed fever four weeks after arrival. Defervescence followed treatment with oral chloroquine 25 mg/kg base for three days. Over the next five weeks he felt tired with anorexia, weight loss of 10 kg, and a dull pain in the right hypochondrium. Liver function tests showed a raised serum bilirubin concentration (38  $\mu\text{mol/l}$ ) and increased serum aspartate transaminase activity (113 IU/l). Viral hepatitis was diagnosed but he continued to deteriorate and was admitted to hospital. On examination he was pale with a body temperature of 36°C, pulse 98/min, and blood pressure 90/60 mm Hg. There was tenderness in the right hypochondrium. Haemoglobin concentration was 106 g/l. A thick blood film showed scanty asexual forms and numerous gametocytes of *P falciparum*. Full clinical and haematological recovery followed treatment with quinine and pyrimethamine-sulfadoxine.

### Comment

None of our patients had fever documented in hospital and none gave a history of fever in the few days before admission. Two patients showed alteration in consciousness and all three patients had hypotension. The first patient, who developed circulatory failure with shock, coma, and oliguria, had the classical features of algid malaria,<sup>2</sup> which because it is uncommon often leads to misdiagnosis.

In 1984 nearly 2000 cases of malaria were recorded in England and Wales, including over 700 due to *P falciparum*<sup>3</sup>. Six of the patients with falciparum malaria died. With the increasing spread of drug resistant strains of this parasite and the continued rise in tourism to countries where malaria is endemic doctors in Britain and Europe seem likely to see ever more patients with this dangerous infection. Delays in diagnosis and treatment of a non-immune person with *P falciparum* may lead to fulminating parasitaemia and risk of death. The absence of fever must not point the doctor away from the diagnosis, and we believe that it is prudent to perform blood smears in any person who develops a febrile or non-febrile illness after visiting a country with endemic malaria.

### References

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- 3 Peters W, Hall AP. The treatment of severe falciparum malaria. *Br Med J* 1985;291:1146-7.

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