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Malaria complicating open-heart surgery

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ABSTRACT Two cases of malaria developing immediately after open-heart surgery are reported to illustrate that malaria is one of the rarer causes of postoperative pyrexia. The diagnosis can easily be missed, resulting in unnecessary morbidity and even mortality. It is important for cardiac surgeons to be aware of this possibility in malaria-free as well as malaria-endemic areas as patients or blood donors who come from or have recently visited an endemic area are potential victims or sources of the infection.

Pyrexia is not uncommon after open-heart surgery. When it persists and the aetiology remains obscure, the important differential diagnoses are infective endocarditis and the postpericardiotomy syndrome. Since the early definitive diagnosis of either condition may not be possible, tentative treatment for these conditions may be instituted in clinical practice as a therapeutic test. However, in rare instances, when an unusual infection such as malaria is responsible for the fever, the diagnosis can easily be missed resulting in unnecessary morbidity and perhaps mortality. This communication describes two cases of malaria immediately after open-heart surgery. Only three similar reports have been published.[1-3] It is important for cardiac surgeons to be aware of this possibility in malaria-free as well as malaria-endemic areas, as patients or blood donors who come from or have recently visited an endemic area are potential victims or sources of infection.[4]

Case reports

CASE 1

This 8½-year-old local Chinese boy (YSH) had closure of a ventricular septal defect on 10 September 1974. Two units of blood were used during the operation and four more units were transfused in the immediate postoperative period. Apart from a persistent fever of 38·2°C his immediate postoperative course was uneventful. On the tenth day, he developed a dry cough and complained of dysuria. Sputum and midstream urine cultures initially yielded *PS aeruginosa*. However, repeated cultures were sterile. Both the cough and dysuria resolved without treatment. From the second week onwards, the persistent fever become remittent in character, reaching 40°C at times. The patient never complained of chills or rigors. During the third postoperative week, his spleen became palpable and progressively increased in size to 5 cm below the costal margin. His haemoglobin fell from 12 g/dl to 8 g/dl. Repeated white cell counts and erythrocyte sedimentation rates were normal. The Widal tests were negative. Despite the fever, his general condition remained surprisingly good. There was no evidence of pulmonary or systemic embolism, but in view of the persistent remittent fever and splenomegaly, a tentative diagnosis of infective endocarditis was made. A course of soluble penicillin was given after taking six blood samples for culture. One of the six blood cultures grew *str faecalis* and the rest were negative. Ampicillin, gentamicin and carbenicillin were added to the antibiotic regimen. However, the fever continued unabated and became intermittent in a quartan pattern six weeks after the operation. Only then was malaria suspected and blood films examined for malaria parasites. Parasitaemia with *Plasmodium malariae* was diagnosed. He was given amodiaquine for three days and primaquine for two weeks. The fever subsided within a few days and the splenomegaly gradually resolved.

The boy had had many attacks of febrile illness in the past, but there was no previous confirmed attack of malaria. There was no history of malaria or of recent visits to any endemic malarious area in any of the six blood

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donors. Repeated blood films taken from them failed to demonstrate any malaria parasites.

CASE 2

A 6-year-old Chinese girl (YMT) from Sabah, Malaysia had closure of a ventricular septal defect on 9 December 1976. Two units of blood were used during the operation and two more units were transfused in the immediate postoperative period. She stood the operation well and appeared to be recovering satisfactorily despite a low grade fever. On the sixth postoperative day, the fever rose to 40°C and she complained of a dry cough and anorexia. Physical examination showed that her general condition was satisfactory and her cardiorespiratory functions were normal and stable, but she was found to have hepatosplenomegaly. Laboratory investigations including white cell counts, six blood cultures, Widal tests, and blood films for malaria, were negative. Her erythrocyte sedimentation rate was persistently over 100 mm in the first hour and her haemoglobin dropped from 13 g/dl on the sixth postoperative day to 5 g/dl on the tenth day. There was no evidence of glucose-6-phosphate dehydrogenase deficiency. Two units of packed red cells were transfused. The fever persisted between 39·4°C and 40·5°C and the haemolysis continued.

On the thirteenth day, her spleen had extended to the level of the umbilicus, haemoglobinuria appeared, and her condition gradually deteriorated. Although repeated blood films had been taken for examination for malaria parasites since the onset of the febrile illness, it was only then that *Plasmodium falciparum* were observed. She was immediately treated with chloroquine. The fever subsided and her general condition rapidly improved. It took four weeks for her spleen to return to normal size and her haemoglobin to be restored to normal. All the blood donors were traced and their blood were examined for malaria parasites. *Plasmodium falciparum* was found in the blood of the girl's father who was one of the blood donors. One unit of his fresh blood had been given to the girl on the day of operation. On careful questioning, the father gave a history suggestive of malaria treated in Sabah over two years previously. The father was also treated with a course of chloroquine.

Discussion

It is purely coincidental that both these cases of malaria followed repair of ventricular septal defects. In case 1, the transmission of malaria was not apparent. Although blood films from all blood donors were free of malaria parasites, this could not entirely exclude the presence of a carrier donor.[4 5] Hence, it is still possible that in case 1 the malaria was transfusion-induced. On the other hand, the operation might have activated a latent infestation.[3] It is generally accepted that *Plasmodium malariae* has secondary tissue forms in the liver, so that the immunosuppressive effect of surgical stress could result in the release of the exo-erythrocytic schizonts in the liver into the circulation.[6] Malaria has been under control for a number of years in Hong Kong, but sporadic cases still occur. Because of the rarity of this condition, the misleading laboratory findings, and the atypical clinical features in the early stage of the disease, the diagnosis of malaria in case 1 was not made for six weeks.

In case 2, the malaria was obviously transmitted by transfusion of infected blood. As a result of the experience with case 1, malaria was suspected early, but nevertheless it took a week to confirm the diagnosis. In retrospect, the child's clinical course warranted antimalarial therapy before laboratory confirmation. Generally, since antimalarial drugs are relatively harmless and effective, they can be used as a therapeutic test. Alternatively, in order to facilitate early diagnosis, blood donors could be examined as soon as malaria is suspected, since transfusion-induced malaria is comparatively more frequent. If blood films fail to demonstrate the malaria parasites, the fluorescent antibody technique can serve as a reliable screening test.[4]

The clinical features of malaria are variable, but when an intermittent fever in a tertian or quartan manner is present, the diagnosis is obvious. Usually the early diagnosis is made on clinical suspicion and routine laboratory screening for malaria parasites. In case 1, all the early findings were compatible with infective endocarditis until the fever failed to respond to the antibiotic regimen and became quartan in character. However, in case 2, there were some features which were not usually seen in febrile illnesses after open-heart surgery—for example, gross splenomegaly and marked haemolysis. These served as clues to the diagnosis of an unusual pathological condition.

It appears that when malaria complicates cardiac operations or indeed any surgical procedures, apart from recent transmission by infected anopheline mosquitoes, it can be mediated by either transfusion of infected blood or activation

of a latent infestation by the stress of surgery. In clinical practice, we feel that the following four points are worth consideration. (1) To prevent transfusion-induced malaria, potential blood donors who have lived in or visited malaria-endemic areas should be rejected. If they are to donate blood, they should be screened by the fluorescent antibody technique.[4] (2) a preoperative prophylactic course of antimalarial drug should be considered in patients from endemic areas with a history of malaria and possible latent infestation. (3) In the event of obscure postoperative pyrexia, malaria should be considered as one of the differential diagnoses, and blood films should be repeatedly examined for malaria parasites in addition to the other laboratory tests. (4) Where the clinical condition of the patient makes a diagnosis of malaria very probable, it is justifiable to give antimalarial treatment even though parasites have not been demonstrated.

Antimalarial drugs are effective and have very few side effects, but early diagnosis and drug treatment are essential for the prevention of life-threatening complications especially in falciparum malaria. Thus, it is important for cardiac surgeons to be aware that malaria can be a cause of postoperative pyrexia and that patients or blood donors coming from or who have recently visited malaria-endemic areas are potential victims or sources.

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