Letters to the Editor

Falciparum malaria and hypoglycaemia

Sir,

I read with interest the case report of Shalev *et al.*¹ on falciparum malaria-induced hypoglycaemia, and I wish to suggest another possible and important mechanism in the pathogenesis of hypoglycaemia associated with falciparum malaria infection.

Several recent studies have demonstrated elevated serum levels of tumour necrosis factor- α (TNF) in patients with falciparum malaria,^{2–6} where this cytokine was also shown to play an important role in the pathogenesis of this serious and potentially fatal infection.^{2–5,7,8} Furthermore, organ impairment in human falciparum malaria was found to be correlated with the amount of circulatory levels of TNF.^{4,5} On the other hand, overproduction of TNF may induce profound hypoglycaemia.^{9–12} Thus, also elevated circulatory levels of TNF may be responsible for the hypoglycaemia associated with falciparum malaria. The mechanisms by which TNF induces hypoglycaemia are not fully understood.

In this regard, it is reasonable to suggest that the beneficial therapeutic effects of the antimalarial agents in malarial infection may be partially mediated through blockade of TNF release, as recent studies have demonstrated that chloroquine inhibits TNF production.^{13–15}

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Atrial fibrillation induced by ibuprofen overdose

Sir,

Deliberate overdose with non-steroidal anti-inflammatory drugs is relatively common, particularly in the case of ibuprofen which is readily available as an 'over-the-counter' preparation.¹ Effects of overdose may include tachycardia, but dysrhythmias are very rare.^{2–4} The authors report a case of atrial fibrillation, apparently secondary to ibuprofen overdose.

A 35 year old man was investigated in 1989 for episodes of retrosternal chest pain. He smoked five cigarettes per day. Routine blood tests including thyroid function were normal. Electrocardiograms showed no abnormality except for a relatively short PR interval of 0.12 seconds. No delta wave was present. Exercise stress testing showed excellent functional capacity without precipitating chest pain. Twenty-four hour electrocardiogram revealed sinus rhythm with no evidence of spontaneous atrial fibrillation. An echocardiogram showed a slightly increased left ventricular end-diastolic dimension of 5.8 cm, but did not detect any valve abnormality. Doppler echocardiography, however, did suggest minimal mitral regurgitation. Subsequent gastroscopy revealed linear oesophagitis, and his pain settled with antacid treatment.

He presented in November 1991, 3 hours after ingestion of 30 ibuprofen 400 mg tablets and two cans of beer. His pulse was 90/minute regular, blood pressure 150/80 mmHg. He was treated with 30 ml syrup of ipecacuanha, with successful induction of emesis, and pink gastric residue was obtained. Plasma ibuprofen levels were not obtained. Thirty minutes later he developed irregular