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

tachycardia and electrocardiogram confirmed atrial fibrillation with a ventricular rate of 170–180 beats/minute. Blood pressure remained stable. The dysrhythmia persisted and required treatment with amiodarone for 3 days before reverting to sinus rhythm. Serial electrocardiograms, and measurement of cardiac enzymes, urea and electrolytes were normal. There were no long-term sequelae.

Ventricular tachycardia associated with hyperkalaemia has been reported after ibuprofen overdose,³ although tachycardia and hypotension are more consistent features.² This is apparently the first report of atrial fibrillation occurring after an overdose of ibuprofen in a previously healthy individual. The presence of mild mitral regurgitation, and a possible hidden accessory pathway (as suggested by the short PR interval) may, however, have been predisposing factors in this case.

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Cerebellar haemangioblastoma in an octogenarian

Sir,

Sporadic cerebellar haemangioblastoma usually affects middle-aged individuals.¹ We recently excised this tumour in an 87 year old woman with subsequent rehabilitation to independence. The mean age at which this tumour presents is rising because a larger proportion of cases are occurring in older patients.² It is believed that during foetal development, a hamartoma forms in the cerebellum from the choroid plexus in the 4th ventricle. Cystic transformation then gives rise to the tumour.³ Why should such a transformation have occurred in our patient so long after the formation of a congenital lesion? Maher has recently provided an answer that requires an understanding of tumour suppresser genes.² Patients with Von Hippel–Lindau syndrome are heterozygous for a recessive tumour suppresser gene and require a single mutation for the formation of familial cerebellar haemangioblastoma. The sporadic tumour only occurs when two spontaneous mutations are acquired during life. This could statistically occur at any age after birth.

Although rare, cerebellar haemangioblastoma should be considered in the differential diagnosis of non-specific symptoms associated with acute cognitive impairment in old patients like ours. The usual physical signs of nystagmus, ataxia and papilloedema may not be elicited if patients are uncooperative in which case a computed tomographic scan of the head is necessary to exclude the diagnosis. A cystic low density mass is seen usually with an enhancing mural nodule. Cerebellar haemangioblastoma is more likely if papilloedema can be demonstrated as it occurs in up to 90% of cases.⁴ When senile miosis and cataract prevent adequate direct fundoscopy, referral to an ophthalmologist for examination by more sophisticated equipment could be valuable. With advances in neuroanaesthesia and a reduction in operative mortality,⁵ elderly patients should not be excluded from major surgery.

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Buerger's disease in Western India

Sir,

The predominant peripheral vascular disease seen in our busy surgical service is Buerger's disease (thromboangiitis obliterans). In 1988–1991 we treated 62 cases of Buerger's disease, all males below 42 years age (mean age 38 years). A total of 51 cases had peripheral gangrene with intractable pain, while the remaining 11 had non-healing ulcers with severe pain requiring narcotic analgesics in increasing doses. Lumbar sympathectomy (unilateral in 52, bilateral in 10 cases) was performed in all patients. Sympathectomy provided only temporary relief of the symptoms, 59 cases ended up in below-knee amputation, while the remaining two patients had minor amputations of the toes. The mean time interval between the onset of symptoms and the amputation was 22 months, while the mean time interval between sympathectomy and amputation was 6.4 months.