

Parasites and the eye

Sir,

It is a pity that Paula Berdoukas and Peter McCluskey did not mention the malaria parasite in their interesting article (vol 64(12), 2003, p. 743).

Cerebral malaria kills thousands of children every year. Early diagnosis is essential and the ophthalmoscope provides a means of achieving it: the retinal appearances are often diagnostic (Lewallen et al, 1999; NV Beare, personal communication, 2003).

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Lewallen S, Harding SP, Ajewole J et al (1999) A review of the spectrum of clinical ocular findings in *P. falciparum* malaria in African children with a proposed classification and grading system. *Trans Roy Soc Trop Med Hyg* **93**: 619–22

PRHO learning and teaching

Sir,

Jeremy Brown et al's (vol 64(12), 2003, p. 740) excellent research into preregistration house officer (PRHO) training is probably representative of all post-graduate deaneries nationwide. Their results clearly show that our health-care system is largely service oriented.

I disagree, however, that 62.9% of PRHOs taking an active part in ward rounds is positive, considering the alarming inactivity in other aspects of their education. Although the PRHOs felt that they had something to gain and contribute their educational supervisors did not share this view.

Fundamental to this is the perception that house officers (HOs) – both senior and preregistration – are not important to the process of health-care delivery and are expendable and therefore not worth the investment of time and effort. I believe this underlies the poor uptake of courses that train the trainer. The structure of our health-care system confers little direct benefit to consultants and managers for training their HOs.

I propose a published league of HO posts which details hospital and/or

departmental practices on teaching, the number of consultants trained to teach and the views of previous HOs regarding their learning experiences, as well as objective markers such as the HO pass rate in college exams. This could promote a learning culture as highly scoring hospitals will be more actively sought, stirring other hospitals to work hard to reach these targets.

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Ibuprofen-induced eosinophilic pneumonia

Sir,

Eosinophilic lung disease is characterized by pulmonary symptoms or an abnormal chest X-ray accompanied by inflammatory cellular infiltrates in the airways and lung parenchyma containing large numbers of eosinophils (Bain and Flower, 1996). Many drugs reportedly cause pulmonary disease (Cooper et al, 1986a,b), but non-steroidal anti-inflammatory drugs (NSAIDs) are rarely mentioned as potential causes of eosinophilic pneumonia – no cases of ibuprofen-induced pneumonia have been published. The following case of eosinophilic pneumonia is believed to be related to ibuprofen therapy.

A 25-year-old man presented with a 2-week illness characterized by a non-productive cough, fever, pleuritic both-sided chest pain and malaise. He denied any weight loss, haemoptysis, night sweats, or contact with persons known to have tuberculosis. He had not recently travelled. Medications on admission included ibuprofen, which the patient had been taking for 7 days for headache and myalgia.

Physical findings included a temperature of 38.2°C, bibasilar rales, increased tactile fremitus and dullness to percussion in both lower lungs; auscultation revealed wheezing in the same areas. Chest X-ray showed multiple reticulonodular opacifications in both lungs. Peripheral white blood cell count was 14 500/mm³ with 38.5%

eosinophils; erythrocyte sedimentation rate was 16 mm/h. Three morning sputum samples were negative for acid-fast bacilli. All sputum, urine and blood cultures were negative. On day 7, he underwent fiberoptic bronchoscopy with bronchoalveolar lavage (BAL). BAL fluid included an abundance of leukocytes with 32% eosinophils. Other serological findings were within normal limits. Cultures from the BAL sample failed to grow organisms.

The patient had experienced two episodes of similar respiratory symptoms in the previous 3 years. These were self-limited and had been diagnosed as probable viral upper respiratory tract infections. They had been temporally related to ibuprofen therapy, taken sporadically for headache and myalgia. Peripheral eosinophilia had been present with each previous episode.

The patient was treated with prednisone 40 mg/day on day 8 with rapid clinical improvement and decline in eosinophilia. He was discharged home on a tapering dose of prednisone. After 1 month he was completely asymptomatic, and a chest X-ray showed complete clearing of pulmonary infiltrates.

The temporal relationship of this illness to ibuprofen intake makes it the likely cause. Medication should be part of the differential diagnosis of diffuse pulmonary infiltrates, particularly in cases associated with eosinophilia or failure to respond to therapy. Medication history must include over-the-counter drugs as a growing number of NSAIDs are available without prescription.

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Drug-induced pulmonary disease. Part 1: cytotoxic drugs. *Am Rev Respir Dis* **133**: 321–40

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Drug-induced pulmonary disease. Part 2: noncytotoxic drugs. *Am Rev Respir Dis* **133**: 488–505