Related disciplines

Most medical specialties have links with other medical and paramedical disciplines. Details of the services provided by such disciplines can be mentioned in the handbook. We find that such information is usefully gathered by visiting the departments, which can be included in any induction course. The handbook should contain details of the referral information required by the particular professionals concerned. When describing social work and appliances it might be useful to reference *Understanding Benefits*[8] and *More Everyday Aids and Appliances*,[9] which should also be available on the ward when possible.

Conclusions

These are only guidelines and every specialty will have its own requirements. We also emphasise that a departmental handbook should be regarded not as an alternative to in service training but as a support and hopefully a stimulus for further learning. Ideally, it should be distributed to junior doctors before the relevant module and should be available for them to take away when they leave for their next job. We must no longer depend on "osmotic learning" as the sole basis of postgraduate medical education, and we must protect, encourage, and stimulate the enthusiasm of young doctors to continue learning in the everyday life of busy hospital wards.

We thank the junior staff for constructive criticism of the first edition of our handbook; Dr Paul Crook for interest, advice, and support; and the Newcastle Rheumatic Research and Education Fund, which financed the project.

1 Dent THS, Gillard JH, Aarons EJ, Crimlisk HL, Smyth-Pigott PJ. Preregistration house officers in the four Thames regions: I. Survey of education and workload. *BMJ* 1990;**300**:713-6.
2 Baker M. Enhancing the educational content of SHO posts. *BMJ* 1993;**306**: 808-9.
3 Regnard CFB, Davies A. *A guide to symptom relief in advanced cancer.* Manchester: Haigh and Hochland, 1986.
4 Dunn DC, Rawlinson N. *Surgical diagnosis and management—a guide to general surgical care.* London: Blackwell, 1988:83-92.
5 Gill GV, Alberti KGGM. Surgery and diabetes. *Hospital Update* 1989;15: 327-36.
6 Chamberlain DA. Advanced life support. *BMJ* 1989;**299**:446-8.
7 *Procedures in practice.* London: BMJ Publishing Group, 1987.
8 Ennals S. *Understanding benefits.* London: BMJ Publishing Group, 1991.
9 Mulley G, ed. *More everyday aids and appliances.* London: BMJ Publishing Group, 1991.

(Accepted 2 July 1993)

Letter from Brasilia

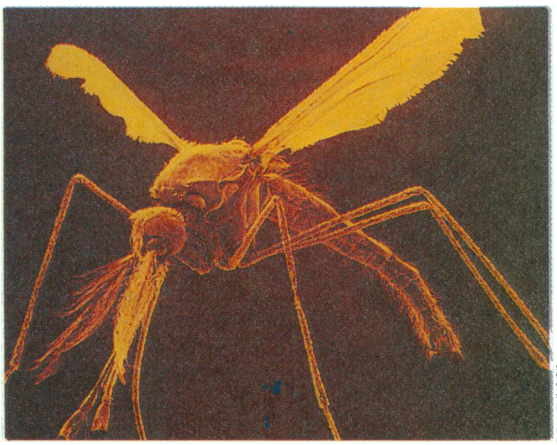
Chronic malaria syndromes

Philip Marsden

Chronic malaria is not a popular concept, especially among older doctors, because of the number of ex-servicemen who claimed compensation for it after the second world war. One of the most convincing temperature charts of 48 hour cyclical fever I have seen was that of a man who had not left London for 10 years. He had his first bout of "malaria" while cleaning the Albert Memorial. The eventual diagnosis was colonic carcinoma with secondary pyogenic emboli. As in this man's experience, once you have known malaria's classic sequence of chills, high fever, and drenching sweats it is natural to think of its return, especially since everybody knows that it is a relapsing infection. Of the four types of human malaria, *Plasmodium falciparum* can relapse for up to two years in an immune subject where initial treatment has been inadequate, and *P vivax* and *P ovale* for up to five. *P malariae* persists latent in the circulation for decades, which is why it figures prominently in the literature of transfusion malaria.

I remember analysing case notes of patients admitted to the Hospital for Tropical Diseases, London, in the early 1960s with an initial diagnosis of malaria. The two commonest causes were dental root sepsis and urinary tract infection. Today, with reliable serological tests, the possibility of occult malaria can be easily excluded.

I have discussed acute malaria in an earlier letter.[1] I wish briefly to discuss here the chronic malaria syndromes found in areas of hyperendemic or holoendemic malaria transmission. I always thought, in my years in sub-Saharan Africa, that these syndromes posed a great clinical problem for the overworked physician. Here, even in Amazonian Brazil, malaria transmission is usually hypoendemic or mesoendemic. Only when you penetrate up small rivers do you encounter higher rates of transmission. I remember sitting with a colleague on the bank of such a river. He was wearing only a pair of shorts, and his legs, arms, and trunk appeared spiny because of the female



BRAIN/SPL

Anopheles gambiae *invaded Brazil from Africa before the second world war; had it not been eradicated, would Brasília have been built?*

anopheline mosquitos standing on their heads to suck his blood. He wanted to take a strain of vivax back and would leave Brazil in time to get his first fever in London.

That situation resembles west Africa and the Gambia, where I spent my first long assignment at the Medical Research Council laboratories doing one of the initial studies of African child health. The Gambia is really a vast swamp. When I entered my house, usually after sunset, a hundred *Anopheles gambiae* mosquitos came in with me through the double screened doors. I dined with them and had to clear my mosquito net before sleep was possible. I took regular effective chemoprophylaxis and never had malaria, although later I got vivax in the New Guinea interior.

In the Gambia, where you are bitten by an infected anopheline every day, I saw how malaria influenced the growth and development of children. They were persistently anaemic, and my laboratory, examining 20-30 slides a day, constantly recorded falciparum

Nucleo Tropical, Universidade de Brasília, CEP 70910, Brasília DF, Brazil
Philip Marsden, *professor*

BMJ 1993;307:555-6

parasitaemias, often asymptomatic, as the children grew and developed their immunity. Without an understanding of malarial immunity and its relatively slow development the clinician is lost in such a locality. Falciparum malaria swamped the *P ovale* and *P malariae* infections also found in west Africa. Nearly every child had clinical falciparum malaria during the first 18 months of life, which resolved with 12·5 mg of pyrimethamine in those days. The development of solid immunity is essential to survival, so radical treatment is not indicated. (I saw Gambian adults with clinical falciparum malaria in only two circumstances; during pregnancy, due to the placenta draining the mother's gammaglobulins, and in people who took antimalaria drugs intermittently, which lowered their natural immunity.) Yanomami Indian children must confront a similar problem in Brazil.

I saw the second chronic malaria syndrome on the other side of Africa, at the New Mulago Hospital in Kampala, Uganda. Today it is called hyperimmune malarious splenomegaly and is characterised by marked hepatosplenomegaly, raised concentrations of IgM immunoglobulins and malaria fluorescent antibody titres, and hepatic sinusoidal lymphocytosis. Our initial report appeared in this journal.[2] In Brazil we first localised this syndrome among bank dwellers on the River Ituxi, a tributary of the Purus River. In this study and in studies from Uganda and New Guinea the syndrome showed a familial tendency, and it has been linked to group DR2 histocompatibility antigen in New Guinea. Usually the syndrome resolves with a prolonged course of antimalarial treatment.

There is much evidence that Burkitt's tumour, the commonest childhood malignancy in Kampala, is the result of Epstein-Barr virus acting on a lymphatic system that has been activated by malaria infection. Recently a physician told me that he had two histologically proved cases from the remote Amazonian state of Acre. Curiously, quartan nephrosis, which was so extensively studied by Giglioli in Guyana, has yet to be reported from Brazil. Foci of *P malariae* are present and it will only be a matter of time before we see reports of nephrotic syndrome due to the specific malarial immunoglobulin damaging the glomerular basement membrane.[3]

Unfortunately our growing knowledge of these subtle syndromes has not been accompanied by equal progress in malaria control. *A gambiae* invaded Brazil from Africa before the second world war but was fortunately eradicated by the old vector control measures. Otherwise—so I teach, at least—Brasília would never have been built. Progress with a malaria vaccine is slow. For this decade we will have to rely on vector control (larvicideal and larviparous fish, insecticide spraying, mosquito nets impregnated with pyrethroid, and chemoprophylaxis.

1 Marsden PD. Growing problem of malaria. *BMJ* 1989;299:1328-9.
2 Marsden PD, Hutt MSR, Wilks NE, Voller A, Blackman V, Shah KK, *et al.* An investigation of tropical splenomegaly at Mulago Hospital, Kampala, Uganda. *BMJ* 1965;i:89-99.
3 Marsden PD. Chronic malaria syndromes and Brasil. *Rev Soc Bras Med Trop* 1990;23:193-6.

Lesson of the Week

Lesions of schistosomiasis mimicking warts on the vulva

P C Goldsmith, T A Leslie, V Sams, A D B Bryceson, E Allason-Jones, Pauline M Dowd

Schistosomiasis may present as warty lesions on the vulva. Take a travel history and a biopsy to make the diagnosis

Schistosomiasis (bilharziasis) is a chronic trematode infection affecting at least 200 million people world wide. Schistosomiasis due to *Schistosoma haematobium* is endemic in Egypt and occurs in most parts of Africa and the Middle East, areas now much favoured by tourists. Infection occurs after bathing in infected fresh water, and symptoms may not appear until many months—sometimes years—after exposure to the parasite. Schistosomiasis can present with lesions on the genitalia that may be mistaken for viral warts. In patients presenting with atypical warts a travel history and biopsy are important as early treatment of schistosomiasis is effective and prevents the serious long term complications of inflammation and fibrosis of the urinary tract.

In this paper we describe three patients living in the United Kingdom who presented with pruritic papules on the vulva. Schistosomiasis was diagnosed on biopsy, and there was no other clinical evidence of infection. On direct questioning all three gave a history of swimming in fresh water in Africa.

Case histories

The first patient was a 29 year old woman who initially presented to a genitourinary medicine clinic with a six month history of an itchy patch on the vulva; she was otherwise well. On examination she had a discrete raised lesion on the left labium minus (fig 1). Results of an infection screen including microscopy and culture for yeasts were negative. Punch biopsy of the lesion under local anaesthetic showed granuloma formation and schistosoma ova with the characteristic terminal spines (fig 2). Investigations revealed a normal blood eosinophil count, a serum IgE concentration of 470 kU/1 (normal 0-120 kU/l), and a positive result on the schistosoma enzyme linked immunosorbent assay (ELISA) against soluble egg antigen. On direct questioning she said that she had swum daily for three weeks in Lake Malawi while on holiday 18 months before.

The second patient was a 26 year old woman who was referred to the gynaecologist with a two month history of an itchy wart on the vulva. Examination showed a 1·5 cm papillomatous area on the right

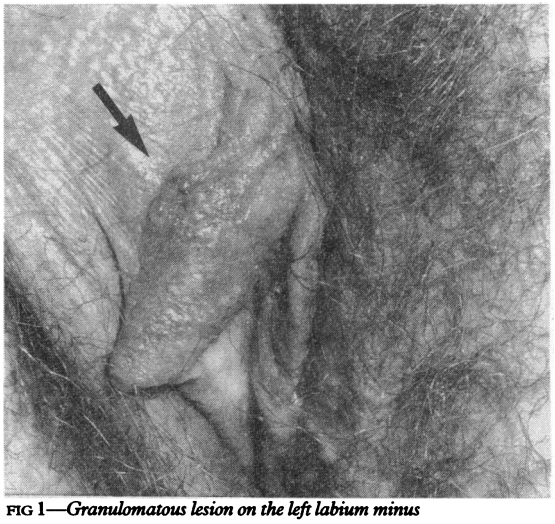


FIG 1—*Granulomatous lesion on the left labium minus*

Correspondence to:
Dr Dowd.

BMJ 1993;307:556-7