Contamination of paraphernalia used for injection—Four addicts submitted their injection paraphernalia for culture. *C. albicans* was isolated from one set and *C. parapsilosis* from another. The remaining two sets yielded negative results.

**Comment**

It has been suggested that juice from fresh lemons may provide a reservoir for candidal infection. To our knowledge, however, plastic lemons have not previously been incriminated. This study suggests that the juice in plastic lemons from which the preservative, sulphur dioxide, has evaporated can become contaminated with *C. albicans*, probably from the addict's flora. Although legal restrictions precluded us from analysing heroin sold in the street for fungal contamination, the fact that diacetylmorphine was found to be fungicidal for *C. albicans* suggests that the heroin itself was a less likely source of infection. Fungal endophthalmitis is a blinding disease and is one manifestation of the multifocal candidal disease that has recently been reported in heroin addicts. We maintain that this additional risk of heroin addiction should be made more widely known as a disincentive to tentative abusers of heroin.

We thank the clinicians of the Glasgow eye departments for making available specimens from their cases and the technical staff of the medical mycology unit for their help.


(Accepted 17 February 1986)

Medical Mycology Unit, Department of Dermatology, University of Glasgow, G1 6NU
G S SHANKLAND, BSc, research assistant
M D RICHARDSON, BSc, PhD, principal mycologist

Tennent Institute of Ophthalmology, University of Glasgow, G11 6NU
G N DUTTON, MD, FRCS, senior lecturer

Correspondence to: Mrs Shankland.

---

**Erythroderma resembling Sézary syndrome after treatment with Fansidar and chloroquine**

There have been reports of serious and indeed fatal multisystem illnesses after patients have received pyrimethamine and sulfadiazine (Fansidar) as malarial prophylaxis for *Plasmodium falciparum* resistant to chloroquine. We describe a patient who developed an illness resembling Sézary syndrome after treatment with chloroquine and Fansidar.

**Case report**

A previously well 45 year old entomologist with no history of allergy visited a northern province of Sumatra to conduct a mosquito survey in an area where chloroquine resistant *falciparum* malaria had been reported. He took chloroquine 150 mg every third day (seven tablets in total) and Fansidar (sulfadiazine 500 mg and pyrimethamine 25 mg) one tablet weekly (six tablets in total).

Three weeks after arrival he became unwell with fever, diarrhoea, and general malaise and was advised to take an extra two tablets of Fansidar. His condition deteriorated, and he arrived in Holland with fever, erythroderma, diarrhoea, and jaundice. There was generalised lymphadenopathy, hepatosplenomegaly with raised activities of hepatic enzymes, and eosinophilia with negative parasitology and viral studies. Treatment was started with prednisolone 40 mg daily, and although the hepatitis improved his general condition remained poor.

On returning to England he was erythrodermic with generalised lymphadenopathy. There was an eosinophilia and a blood film showed abnormal lymphoid cells with features of Sézary cells. The following yielded normal results: blood count; measurement of erythrocyte sedimentation rate, Paul-Bunnell, Venereal Disease Research Laboratory, and *Treponema pallidum* haemagglutination test; tests for hepatitis B surface antigen, hepatitis A antibodies, and human
T cell lymphotropic virus type III antibodies, filaria and schistosomal enzyme-linked immunosorbent assay; testing of stools for ova and parasites; tests of liver function and autoantibodies; chest radiography; and abdominal ultrasound. Immunoglobulin concentrations were normal apart from a slightly raised IgM. Skin biopsies on admission showed focal parakeratosis with acanthosis of the epidermis, in which numerous convoluted lymphocytes were identified. A repeat skin biopsy taken three weeks later when the skin had improved showed a perivascular lymphocytic infiltrate. Biopsy of the lymph nodes was consistent with a dermatopathic lymphadenitis.

The erythromedra improved, but the patient still required 5 mg prednisolone on alternate days and a moderately potent (group III) topical steroid cream five months after onset of illness.

**Comment**

From 1982 to 1985 between 109 000 and 156 000 people were exposed to Fansidar in the United States. Twenty severe cutaneous reactions (erythema multiforme, Stevens-Johnson syndrome, and toxic epidermal necrolysis) have been documented, 19 of which occurred in people using chloroquine simultaneously. Six of these reactions were fatal. These reactions were associated with multiple (two to five) doses of Fansidar only when used as weekly prophylaxis, and none of the serious reactions were associated with treatment with single doses of Fansidar, as used to treat malaria.

**Plasmodium falciparum** resistant to chloroquine is an increasing problem in malarial areas. Fansidar has been widely recommended for prophylaxis, but awareness of its potentially serious side effects has grown, and the combination of Fansidar with chloroquine, as in our patient, seems particularly liable to cause serious reactions.

We thank Dr F Allenby (consultant dermatologist, Lister Hospital, Stevenage) for referring the patient and Dr J Thompson (chef de clinique, Academisch Ziekenhuis, Leiden, Holland). We acknowledge the help and advice of Dr A Bryceson (Hospital for Tropical Diseases, London) and Dr P W M Copeman (consultant dermatologist, St Stephen’s Hospital, London).


(Accepted 28 February 1986.)

---

**Toxoplasmosis in cardiac transplantation**

Despite new immunosuppressive regimens infections with opportunistic organisms still constitute an important threat to patients undergoing organ transplantation. Toxoplasma gondii may cause fulminating and rapidly fatal infection in recipients of heart transplants. The report of prevalence of infection with T gondii in recipients of heart transplants at Papworth Hospital, the role of the donated hearts as a source of infection, successful treatment of fulminant infections, and the role of pyrimethamine in prophylaxis.

**Patients, methods, and results**

Altogether 119 patients who had received cardiac or cardiopulmonary transplants were reviewed (106 men, 13 women; age range 9-54 (mean 38.6) years).

Seventeen patients received conventional immunosuppression with azathioprine and steroids; cyclosporin A was used with low dose steroids in 80 patients and with azathioprine in 22. All patients received a short course of intravenous antihymocyte globulin.

Infection due to T gondii was diagnosed if a fourfold or greater rise in the late agglutination antibody titre was confirmed by a similar rise in dye test titre or the finding of cysts of T gondii in myocardial biopsy specimens, or both. The postoperative follow up period ranged from three to 72 months. Patients who developed infection with T gondii were treated with oral pyrimethamine 25 mg twice daily and spiramycin 1 g twice daily. In addition, sulfadiazine 1 g was given initially four times daily intravenously and replaced later with a mixture of three sulphonamides (Sulphatriad: sulphadiazine 185 mg, sulpha-merazine 130 mg, and sulfapheazoate 185 mg; May and Baker Ltd) three times daily. After the acute phase of the illness treatment continued with pyrimethamine and Sulphadiazine for 10-12 months. As our initial experience showed that infection with T gondii was most likely to occur in seronegative recipients of hearts from seropositive donors we later used pyrimethamine for prophylaxis in this group.

Pyrimethamine was administered as a single daily dose of 25 mg for six weeks postoperatively.

Results in seronegative recipients of hearts from seropositive donors (n = 14)---Early in the series seven patients did not receive prophylactic pyrimethamine. Four of these developed clinically overt infections with T gondii 20-32 days after transplantation (mean 26 days). Two patients died, but the other two were successfully treated. In these four patients cysts were seen in the myocardium on biopsy in three patients and at necropsy in one. Of the patients who developed toxoplasmosis, one was given conventional immunosuppression and three cyclosporin A. Later in the series seven patients received pyrimethamine as prophylaxis; none developed primary infection with T gondii.

Results in seronegative recipients of hearts from seronegative donors (n = 66)---None of these patients developed toxoplasmosis.

Results in seropositive recipients (n = 39)---One patient developed a clinically mild reactivated form of infection with T gondii with an appreciable rise in antibody titre. He was treated successfully.

**Comment**

We believe that our success in managing patients with disseminated toxoplasmosis was due to early diagnosis and treatment. The use of spiramycin in addition to conventional treatment with pyrimethamine and sulphonamides may also have contributed. Cyclosporin A inhibits replication of T gondii in vitro at high concentrations. Conceivably, therefore, the peak concentrations in the blood and tissues may exert an antitoxoplasma effect in vivo.

Because primary toxoplasmosis occurred only in the "mismatch" group, in which seronegative patients received hearts from seropositive donors, we consider that the presence of T gondii antibody in a donor indicates the presence of T gondii in the donor’s heart. To identify this high risk group it is necessary to screen both recipients and donors for T gondii antibody. The ideal would then be to ensure that organs from seropositive donors are not transplanted into seronegative recipients. This, however, is not practicable. Our preliminary experience with prophylaxis with pyrimethamine suggests that it confers considerable benefit, although more data are required to reach a firm conclusion at conventional levels of significance.


(Accepted 19 February 1986.)

---

**Department of Cardiothoracic Surgery, Papworth Hospital, Cambridge CB3 8RE**

M HAKIM, FRCS, MRCP, registrar in cardiothoracic surgery

D ESMORE, FRCS, registrar in cardiothoracic surgery

J WALLWORK, BSC, FRCS, consultant cardiothoracic surgeon

A H ENGLISH, BSC, FRCS, consultant cardiothoracic surgeon

Public Health Laboratory, Addenbrooke’s Hospital, Cambridge CB3 8RE

T WREGHITT, PHD, MRCPATH, principal microbiologist

Correspondence to: Mr Hakim.