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.

Recovery of fungi from street heroin—It is illegal to obtain heroin sold in the streets. Legal restrictions on the availability of confiscated drugs precluded analysis, despite our attempts to arrange this.

Effect of diacetylmorphine hydrochloride on growth of *C. albicans*—Previous studies failed to isolate *Candida* spp from heroin sold in the street.^{2,3} Cultures of *C. albicans* in the exponentially growing and resting phases were found to be inhibited by diacetylmorphine in agar diffusion susceptibility tests. Excision of the inhibition zones and further incubation in drug free medium did not enhance growth of the organisms. This indicates a fungicidal potential for the drug. The minimum inhibitory concentration using agar and broth dilution methods was 25-30 g/l.

Comment

It has been suggested that juice from fresh lemons may provide a reservoir for candidal infection.^{4,5} 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.² 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.

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Autoamputation of infant's finger by knitted mitten: a forgotten hazard

It is nearly 20 years since the British Medical Association drew attention to the danger to infants' fingers posed by mittens knitted from synthetic materials.¹ The case described below is thus a timely reminder.

Case report

A 5 week old girl was put to sleep in knitted mittens by her parents. Overnight the infant was fretful, but the parents did not realise why. In the morning they could not remove the left mitten and so took her to their general practitioner. He carefully cut off the mitten to find the tip of the index finger swollen and black (figure).

Dry gangrene had been caused by the finger passing through the open knitting of the mitten and becoming stuck, so that the blood supply had been cut off. An expectant policy was adopted; the blackened finger tip was demarcated and separated, and the remaining stump healed satisfactorily with the loss of the terminal phalanx.

Swollen and black index finger.

Comment

The incidence of similar unfortunate episodes is unknown. Review of Hospital Activity Analysis data listed under "amputations" for the years 1975-84 shows that less than 1% of amputations occur among infants under 1 year of age; most occur in men aged over 20 and are probably due to accidents at work. The amputations listed are almost certainly surgical procedures, and cases such as that above are probably not included.

This case is important because it could have been prevented. Clothing for infants is often knitted, and is either produced by commercial manufacturers or home made. Ornamental knitting patterns with fairly large holes and the use of less yielding synthetic fibres increase the risk of digits becoming trapped between strands. It would be safest to use only tight woven rather than knitted material for infants under 1 year of age.

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Erythroderma resembling Sézary syndrome after treatment with Fansidar and chloroquine

There have been reports of serious¹ and indeed fatal² multisystem illnesses after patients have received pyrimethamine and sulfadoxine (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 (sulfadoxine 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 hepatocellular 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 tests; 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 erythroderma 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.¹

Plasmodium falciparum resistant to chloroquine is an increasing problem in malarial areas. Fansidar¹ 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.⁵

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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 fulminant and rapidly fatal infection in recipients of heart transplants.¹ We report the 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 antithymocyte globulin.

Infection due to *T gondii* was diagnosed if a fourfold or greater rise in the latex agglutination antibody titre was confirmed by a similar rise in dye test titres or the finding of cysts of *T gondii* in myocardial biopsy specimens, or both.² 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, sulphadiazine 1 g was given initially four times daily intravenously and replaced later with a mixture of three sulphonamides (Sulphatriad: sulphadiazine 185 mg, sulphamerazine 130 mg, and sulphathiazole 185 mg; May and Baker Ltd) three times daily. After the acute phase of the illness treatment continued with pyrimethamine and Sulphatriad 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.³ 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 two others 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 titres. 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.⁴ Conceivably, therefore, the peak concentrations in the blood and tissues may exert an antitoxoplasma effect in vivo.⁵

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 probable presence of *T gondii* cysts 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.

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