the study were grouped according to their length of stay in the unit. The prevalence of the outbreak strain among patients who spent less than three days in the unit was 0·85% (2/235 patients), compared with 54% (32/59) among those who stayed in the unit for more than three days. Among this second group, the 27 patients who never carried the outbreak strain spent an average of 8·6 days in the unit (total 233 days) whereas the 32 patients who did acquire the outbreak strain spent an average of 21 days in the unit (total 673 days). Thus there was a higher risk of colonisation by the outbreak strain for patients who remained in the unit for a long time. The average time between admission to the unit and colonisation with the outbreak strain was 6·6 days. This was consistent with a model of superinfection with an exogenous candidal strain. Four of the patients who subsequently died from infection with the outbreak strain were initially colonised by a different candidal phenotype. In two cases this was *C tropicalis*. These patients did not develop systemic disease from their original endogenous yeast but became superinfected by the endemic outbreak strain, which then disseminated.

The outbreak was due to a particular strain of *C albicans* (serotype A, morphotype A1, biotype $0/_{1}5\ 5/_{7}$). This strain survived better on nurses' hands than control strains of *C albicans* and was considerably more resistant to washing with Hibiscrub, the disinfectant mainly in use at the time of the study. The lack of an environmental source agrees with earlier work[20 21] and accords with the observation that the outbreak strain did not survive better on blocks of formica than the control strains.

Of the 65 staff examined, four were oral carriers of the outbreak strain and one carried this strain on her hands. While nursing patients with systemic infections two out of 17 nurses acquired the outbreak strain. One nurse developed clinical vaginal candidosis 48 hours after nursing a patient (case 15). She had no history of the disease, and the isolate was the same as the outbreak strain. The data suggest that the cycle of infection lies between patients and staff.

In conclusion, we found that one particular strain of *C albicans* was capable of causing systemic candidosis as a result of cross infection between patients and staff. It not only caused invasive infections but showed a propensity to spread, possibly due to its relative resistance to washing with Hibiscrub. Previously, chemoprophylaxis has concentrated on eradicating endogenous yeast flora from the gastrointestinal tract.[3] This should perhaps be combined with isolation of patients with systemic infections and heavy colonisation as well as handwashing with disinfectants that are active against candida.

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(*Accepted 28 December 1984*)

SHORT REPORTS

Prolonged hypercalcaemia after industrial exposure to vitamin D_3

Hypercalcaemia due to vitamin D intoxication is usually the result of a prolonged oral intake or overdosage. Individual tolerance may vary and occasionally patients may be sensitive to low doses. We report a case in which brief exposure to a process manufacturing vitamin D_3 (cholecalciferol) led to prolonged intoxication and hypercalcaemia.

Case report

A previously fit 32 year old laboratory technician presented on 21 April 1983 with a three week history of increasing polydipsia, anorexia, nausea, and general malaise. He had no medical history and was not taking any medication. He had helped in the crystallisation of vitamin D_3 for 32 days in 1981, 11 days in 1982, and 22 days in 1983. Symptoms had started two days after he had last begun working on the manufacture of vitamin D_3. There had been no other relevant exposure. A dust mask, laboratory coat, and gloves had been worn during all the procedures. Examination showed a fit, lean young man who had signs of mild dehydration. His blood pressure was 120/80 mm Hg with no postural drop and he was clinically normal.

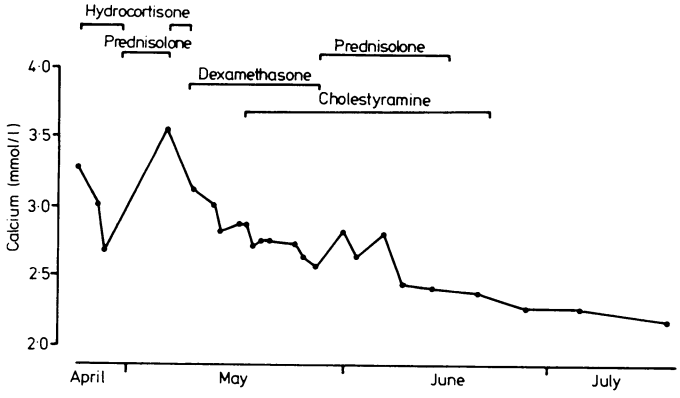
The initial serum calcium concentration was 3·5 mmol/l (14·0 mg/100 ml) (normal 2·2-2·7 mmol/l), phosphate 1·06 mmol/l (3·3 mg/100 ml) (normal 0·8-1·45 mmol/l), alkaline phosphatase activity 154 U/l (8 KA units/100 ml) (normal 100 U/l), and blood urea concentration 9·1 mmol/l (55 mg/100 ml) (normal 2·5-7·5 mmol/l). Serum creatinine concentration was 170 μmol/l (1·9 mg/100 ml) (normal 30-170 μmol/l). The chest radiograph was normal and abdominal radiography showed no evidence of nephrocalcinosis. The parathyroid hormone concentration was less than 0·1 μg/l (normal less than 0·1-0·73).

The patient was treated with intravenous saline, frusemide 40 mg eight hourly, and hydrocortisone 200 mg six hourly. His general condition improved and the calcium concentration dropped to 2·5 mmol/l (10·0 mg/100 ml) after 10 days of treatment. He was discharged and continued with prednisolone 30 mg daily and a high fluid intake. He was readmitted a week later with a calcium concentration of 3·7 mmol/l (14·8 mg/100 ml), and despite adequate rehydration, low calcium diet, and steroids the calcium value remained high and widely fluctuant. He developed a gross Cushingoid appearance and hence the dose of hydrocortisone was gradually reduced over one week and replaced with dexamethasone, which has less tendency to cause fluid retention. Two weeks later he began cholestyramine 8 g twice daily, and over three weeks the calcium concentration fell, fluctuating between 2·5 and 2·85 mmol/l (10·0 and 11·4 mg/100 ml). After eight weeks' treatment his calcium concentrations were normal and remained so with a normal diet and no treatment (figure). After the diagnosis there was no further exposure to vitamin D_3. The initial concentrations of 25-hydroxycholecalciferol one month after the exposure to vitamin D_3 were grossly

raised at 496 ng/ml (dilution 1/5) and 471 ng/ml (dilution 1/10) (normal 3-30 ng/ml). Subsequent estimations on 13 September and 24 November 1983—that is, six and eight months after exposure—remained raised at 139 and 116 ng/ml respectively.

Comment

Vitamin D_3 intoxication in an industrial setting has not been reported before. The usual route of intoxication is oral.[1] In our patient the route of intoxication is unknown. During the 22 days of exposure before presentation the technique of manufacture was changed so that, whereas the solution of vitamin D_3 was previously cooled in a two stage procedure to 4°C overnight and then to −10°C, the new procedure entailed rapid cooling to −10°C. This produced finer crystals than before, and possibly these were inhaled. The rapid onset of symptoms within a few days of exposure and the gross and persistent increase of vitamin D concentrations after such brief exposure are remarkable.



Serum calcium concentrations and treatment during 1983.
Conversion: SI to traditional units—Calcium: 1 mmol/l≈4 mg/100 ml.

Oral preparations of vitamin D_2 (ergocalciferol) and vitamin D_3 (cholecalciferol) are absorbed from the small intestine. Most of the vitamin appears first in the lymph and principally in the chylomicron fraction. Bile is essential for adequate intestinal absorption. The absorbed vitamin D circulates in the blood in association with vitamin D binding protein, which is a specific γ globulin. Vitamin D is stored for prolonged periods in fat deposits throughout the body and is primarily excreted in the bile and partly reabsorbed as a component of the enterohepatic circulation; a small amount is found in the urine.[2]

The clinical effectiveness of glucocorticoids in the treatment of vitamin D intoxication has been attributed primarily to reduction of intestinal absorption of calcium and to inhibition of bone resorption.[3] Altered vitamin D metabolism has been suggested by some studies. In this case, however, the hypercalcaemia persisted despite adequate glucocorticoid treatment, and the bile salt binding agent cholestyramine was therefore used to enhance vitamin D excretion.[4] We could not evaluate this treatment owing to the lack of serial vitamin D concentrations before and during cholestyramine. The calcium concentrations, however, showed a more sustained reduction with cholestyramine compared with the previous prolonged use of high dose corticosteroids (figure).

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Blood donors at high risk of transmitting the acquired immune deficiency syndrome

The acquired immune deficiency syndrome (AIDS) occurs most commonly in homosexual men.[1] This group carries the greatest risk of transmitting AIDS by blood transfusion. Curran *et al* implicated transfusion of blood and its derivatives as a potential mode of transmission of AIDS[2]; transfusion services are trying to minimise this risk. Initially, in the absence of an identified transmissible agent, tests for antihepatitis B core antigen and *Treponema pallidum* haemagglutination were proposed for detection of donors at a high risk of transmitting AIDS. A more direct approach is to defer those donors who are more likely to contract the disease by asking them not to give blood at that particular session. A leaflet describing the importance of AIDS and listing high risk groups was prepared by the Department of Health and Social Security; it implied that only promiscuous homosexuals should voluntarily exclude themselves from giving blood. Subsequently the leaflet was revised to state that this advice applied also to non-promiscuous male homosexuals.

Subjects, methods, and results

Despite the leaflet, at our transfusion centre some male homosexuals still gave blood. Up to 1983 about eight of the 50-60 donors found each year to carry hepatitis B surface antigen had acute hepatitis B virus infections. In early 1984 nine acutely infected donors (mostly young white male homosexuals) accounted for 36% of the donors who carried hepatitis B surface antigen. This prompted us in July 1984 to give a questionnaire to all donors attending a blood donor clinic in the west end of London. At this clinic 87% of donors had given blood previously and 53% were male. Donors were given a leaflet on AIDS (revised at our centre to include all practising male homosexuals in the high risk category) and a questionnaire to complete in private. Those who considered themselves to be in a high risk group were asked to designate their blood for research purposes only. These donors were subsequently interviewed in private by a medical officer. Serum samples from donors who confirmed that they were in the high risk category were tested for antihepatitis B core antigen[3] and anti-human T lymphotropic virus type III (anti-HTLV-III)[4] in addition to the routine screening of donors for hepatitis B surface antigen and syphilis.

All donors who belonged to a high risk group were men. Homosexuality was the only risk factor, and none admitted to intravenous drug abuse. Of 5000 questionnaires administered between July and October, 614 were not completed or had ambiguous answers. The table summarises the results. Overall, 38 donors who completed the questionnaire belonged to a high risk group. Of these, none were positive for anti-HTLV-III, *T pallidum* haemagglutination, or hepatitis B surface antigen but seven were positive for antihepatitis B core antigen. Of the 2333 men who did not categorise themselves as high risk, one was positive for *T pallidum* haemagglutination and one for hepatitis B surface antigen.

Blood donors at high risk of contracting the acquired immune deficiency syndrome

Blood donor	Total No	No (%) at high risk
Established:		
Male	2105	35 (1·7)
Female	1705	0
First time:		
Male	228	3 (1·3)
Female	348	0
Total	4386	38 (0·9)

Comment

Cheingsong-Popov *et al* found that 59% of symptomatic homosexuals and 42% of their contacts were positive for anti-HTLV-III,[4] but one thousand random blood donors from our centre were negative. Similarly, all 38 donors in our study who admitted homosexuality were negative for hepatitis B surface antigen, *T pallidum* haemagglutination, and anti-HTLV-III; most, however, would have been screened for hepatitis B surface antigen and syphilis at previous donations.

Donors in the high risk category said that they had continued to donate despite the publicity about AIDS because the original