of 10% podophyllin for self-treatment, apparently without any instructions. After a week of twice-daily application to the warts and failing to wash off the podophyllin, she developed severe vulval inflammation which resulted in her calling out an emergency cover GP and subsequently being referred to our department. Secondarily infected vulval ulceration was treated with oral flucloxacillin, simple analgesia and saline baths. In the medium-term follow-up the patient has reported a persistent burning sensation and discomfort in the vulval region which has prevented sexual intercourse.

These cases illustrate some of the potential problems of prescribing podophyllin solutions for self-treatment in female patients. Podophyllin solutions are toxic and can cause chemical burns even when used with due care. Rare but serious systemic effects have been reported, usually following ingestion or application of large volumes of podophyllin to damaged epithelial surfaces; these include possible fetal malformation^{1,2} and intra-uterine death.³ The confusion over the strength of the podophyllin solution in the first case illustrates the hazards of writing prescriptions for non-standardised solutions. Furthermore, podophyllin is not licensed for self-treatment in the UK.

In a small verbal survey which we conducted at a recent scientific conference, of 33 consultant genitourinary physicians questioned, 31 said that they would never prescribe podophyllin for female self-treatment; all cited the difficulties in accurate application and the risk of conception during treatment as their principle reasons. The two physicians who said that they would prescribe podophyllin, did so only with the provisos that there were few, non-mucosal warts and that the patient must be judged to be highly dependable in following instructions for application.

We believe that all patients presenting to their GPs with genital warts should be referred to a department of genitourinary medicine unless the GPs can screen for other sexually transmitted infections, treat, test for cure and contact-trace as appropriate. We would discourage the prescription of podophyllin for self-treatment by female patients. For those women unwilling or unable to attend either a genitourinary medicine clinic or their GP's surgery for treatment, podophyllotoxin 0.5% is a safe, effective alternative.^{4,5}

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- 1 Karol MD, Conner CS, Watanabe AS, Murphrey KS. Podophyllin: suspected teratogenicity from topical application. *Clin Toxicol* 1980;16:283-6.
- 2 Cullis JE. Congenital deformities and herbal 'slimming tablets'. *Lancet* 1962;ii:511-2.
- 3 Chamberlain MJ, Reynolds AL, Yeoman WB. Toxic effect of podophyllin application in pregnancy. *BMJ* 1972;3:391-2.

- 4 Baker DA, Douglas JM Jr, Buntin DM, Micha JP, Beutner KR, Patsner B. Topical podofilox for the treatment of condylomata acuminata in women. *Obstet Gynecol* 1990;76:656-9.
- 5 Greenberg MD, Rutledge LH, Reid R, Berman NR, Precop SL, Elswick RK. A double-blind, randomized trial of 0.5% podofilox and placebo for the treatment of genital warts in women. *Obstet Gynecol* 1991;77:735-9.

Periareolar breast abscess due to *Pseudomonas aeruginosa* in an HIV antibody positive male

Breast abscesses in men are rare and there are no reports of such infection in men who are HIV antibody-positive. We present a case of an HIV-infected man who developed two periareolar abscesses due to infection with *Pseudomonas aeruginosa*. A 33 year old bisexual man presented with a week's history of pain in the left breast. There was no history of nipple-piercing. He had been known to be HIV antibody-positive for 13 months following the diagnosis of oral hairy leukoplakia, oral pseudomembranous candidiasis and perianal herpes simplex, at which time he had a CD4+ lymphocyte count of 85/mm³. His current medication was zidovudine 200mg t.d.s, and co-trimoxazole 960mg o.d.

On examination he was thin and afebrile and there was a small erythematous area above the nipple of the left breast which on palpation was tender, non-fluctuant and indurated. There was no associated lymphadenopathy. Investigations showed a CD4+ lymphocyte count of 43/mm³ and a granulocyte count of $1.6 \times 10^9/l$ (normal 2.0-7.5). A diagnosis of periareolar cellulitis was made and treatment with erythromycin 500 mg b.d. commenced. Three weeks later a 4 cm² abscess developed and malodorous pus was obtained by needle aspiration. Culture of the pus grew *P. aeruginosa* and treatment was changed to ciprofloxacin 500 mg b.d. After approximately five weeks of ciprofloxacin the abscess, which since aspiration had drained freely via the aspiration site, had largely resolved. However, a second periareolar abscess developed, adjacent to the first (fig).

Left nipple and adjacent abscesses.

This was incised and drained under local anaesthetic and the pus cultured grew *P aeruginosa* sensitive to ciprofloxacin.

After 11 weeks' treatment with ciprofloxacin, the skin between the abscesses broke down to reveal a cavity; betadine packs were inserted and necrotic tissue at the wound edges was debrided. After 14 weeks' treatment with ciprofloxacin and abscess cavity packing, swabs taken from the cavity yielded no bacterial growth. Wound care with granu-flex was commenced and treatment with ciprofloxacin continued for a further 5 weeks. The abscesses were completely healed 23 weeks after the patient's initial presentation.

Breast abscesses are common in women but rare in men. To our knowledge there is only one published report of non-lactational breast abscesses in an HIV-positive individual, a woman whose breast abscess was caused by *Mycobacterium tuberculosis*.¹ We believe that ours is the first reported case of breast abscesses in an HIV positive male. Recently, a case of gonococcal mastitis has been reported in a homosexual male who wore a nipple ring, but the HIV status of this patient was not known.²

Periareolar breast abscesses are associated with underlying periductal mastitis³ and heavy cigarette smoking has also been implicated in their aetiology.^{4,5} It is interesting to note that our patient smoked approximately 30 cigarettes a day. Culture of *P aeruginosa* in this case is in keeping with the increased susceptibility to infection with this organism seen in immunosuppressed patients.

Some authors have recommended that successful management of periareolar abscesses necessitates surgical excision of infected tissue, including partial nipple excision where necessary.^{6,7} One of these studies⁶ showed a higher risk of relapse in those cases managed by simple incision and drainage. However, none of these studies related to HIV positive patients. The patient we describe was generally well throughout this period and despite a very low CD4+ count recovered with outpatient treatment. Subsequently, despite a general deterioration in his health in association with a further fall in his CD4+ lymphocyte count, there has been no recurrence of the abscesses.

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1 Hartstein M, Leaf HL. Tuberculosis of the breast as a presenting manifestation of AIDS. *Clin Infect Dis* 1992; 15(4):692-3.

2 Bodsworth NJ, Price R, Nelson MJ. A case of gonococcal mastitis in a male. *Genitourin Med* 1993;69:222-3.

3 Hughes LE, Mansel RE, Webster DJT. The ductal ectasia/periductal mastitis complex. In: Hughes, ed. *Benign disorders and diseases of the breast: Concepts and Clinical Management*. London: Bailliere Tindall 1989.

4 Schafer P, Furrer G, Mermillod B. An association of cigarette smoking with recurrent subareolar breast abscesses. *Int J Epidemiol* 1988 Dec;17(4):810-3.

5 Bundred NJ, Dover MS, Coley S, Morrison JM. Breast abscesses and cigarette smoking. *Br J Surg* 1992;79: 58-9.

6 Watt-Boolsen S, Rasmussen NR, Blichert-Toft M. Primary periareolar breast abscess in the non-lactating breast: risk of recurrence. *Am J Surg* 1987;153(6): 571-3.

Accepted for publication 17 October 1993

Lactic acidosis, non-Hodgkins lymphoma and the acquired immunodeficiency syndrome

Chattha *et al* have recently reported seven cases of patients with the Acquired Immunodeficiency Syndrome (AIDS) who developed severe lactic acidosis in the absence of hypoxaemia, malignancy or other obvious causes.¹ We report a case of AIDS related Burkitts lymphoma complicated by Type B lactic acidosis.

A 26 year old male, diagnosed as Human Immunodeficiency Virus (HIV) antibody positive in 1985 secondary to intravenous drug use, was admitted in November 1992 with a 3 week history of generalised malaise, anorexia, and subjective weight loss. He had a past history of exposure to hepatitis B and C viruses as a result of his drug use. He has not used non-prescribed drugs since the time of diagnosis in 1985, and had not received zidovudine therapy at any stage.

On admission he was afebrile and normotensive. There were no clinical stigmata of chronic liver disease. Abdominal examination revealed splenomegaly and an epigastric mass. Respiratory examination revealed left basal coarse crepitations. Sputum culture grew *Haemophilus influenzae* and *Streptococcal pneumoniae* species but at no stage was the patient clinically septic or shocked. A chest radiograph was normal. Initial investigations pH = 7.23 (7.35-7.45), pO₂ 9.00 kPa (11.00-15.00), pCO₂ 5.46 kPa (4.50-6.00), oxygen saturation 91%, actual bicarbonate 16.0 mmol/l (normal 22-28), standard bicarbonate 16.0 mmol/l (normal 21-27) and base excess 10.0 mmol/l (normal -2.0-2.0). Full blood count was normal. Serum biochemistry showed a urea of 13 mmol/l (normal 3-7) and creatinine of 75 µmol/l (normal 50-125). Urate level was 861 mmol/l (normal 100-450). Liver function tests showed elevation and with some changes from assays in 1989 (table). Lactate dehydrogenase (LDH) level was 1081 IU/l (100-350). The anion gap was calculated at 23 (normal 12-15). Blood glucose was 3.5 mmol/l and toxicology screen for illicit drug use was negative. Lactate levels were recorded elevated at 7.9 mmol/l (normal 0.5-2.2). All other causes of elevated anion gap metabolic acidosis were excluded by appropriate laboratory measures.

Repeat LDH was 3696 IU/l, the subfractionation of which showed predominant LD2 and LD3 isoenzymes, suggestive of lymphoproliferative or myeloproliferative origin. Abdominal ultrasound confirmed liver

Umbilical warts: a new entity?

The two cases reported by Nathan¹ are not a new entity. Anwyl-Davies² described a 19 year old woman who presented with a three week history of vaginal and umbilical discharge. Microscopy revealed the presence of gonococci in both sites. During the following two months a pedunculated tumour developed at the umbilicus and rapidly enlarged. It looked "like a raspberry", and "exactly resembled a vulval wart". Similar lesions developed on the vulva and perineum.

The umbilical tumour failed to respond to topical treatment, and it was excised; healing was rapid. Although histopathology was not performed, it seems likely that this patient had vulval, perineal and umbilical condylomata acuminata.

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1 Nathan M. Umbilical warts: a new entity?
Genitourin Med 1994;70:49-50.

2 Anwyl-Davies T. Gonococcal papilloma at the umbilicus. *BMJ* 1919;1:377-8.

BOOK REVIEW

HIV, AIDS, and the Brain. Edited by Richard W Price and Samuel W Perry. (Pp 352, US\$125, hardback.) New York, Raven Press. 1993. ISBN 0-7817-0063-9.

Although there has been considerable research into the central nervous system manifestations of HIV infection, the exact cause of the dementia and myelopathy remains obscure. There is therefore a need for ongoing dialogue between clinicians and scientists to discuss future directions for research. This volume represents the published proceedings of the December 1992 meeting of the Association for Research in Nervous and Mental Disease (ARNMD). The meeting had the brief of reviewing the current knowledge regarding human immunodeficiency virus (HIV)-associated

CNS disorders and also to point towards future directions for research.

Since the first description of dementia in HIV infected patients (including seminal studies by Dr Price) there has been an intriguing discrepancy between the presence of cognitive impairment and the presence and severity of HIV-related neuropathological changes, such as HIV encephalitis or leukoencephalopathy. Some of the discrepancy is perhaps explained by the recent finding of neuronal loss without evidence of direct neuronal infection. This neuronal loss is presumably mediated through one or more neurotoxins, for which there are several candidates, including viral GP120 and cytokines produced by the association of infected macrophages and neighbouring astrocytes. This book considers these and other basic science issues including HIV-associated neuropathological changes, HIV neurotropism, HIV-glial cell interactions, HIV and cytokine expression, HIV and NMDA receptor-mediated neurotoxicity, and the relationship between central and peripheral manifestations of HIV-associated neurological disorders. The clinical chapters include comprehensive reviews of psychiatric aspects of HIV infection, epidemiology and risk factors for HIV-associated dementia complex, neuropsychological assessment (including CNS monitoring in anti-retroviral drug trials), and encephalopathy of childhood.

The debate about pathogenesis is far from sterile and highlights the therapeutic avenues that will be pursued in the next decade. As patients with AIDS are better protected by prophylaxis against opportunistic infections and by antiviral drugs they will be increasingly exposed to the later complications of AIDS of which dementia is the most feared.

This book should not be seen as a manual of neurological manifestations of HIV infection but as an extremely valuable focused review of the scientific background to the principle neuropsychiatric complications. Perhaps its most useful chapters are those which bookend the volume: the first by Dr Price and the last by Dr R T Johnson. These chapters attempt to synthesise the current knowledge regarding HIV and to consider its place in our current understanding of neurovirology and neuroimmunology.

This volume will be of interest to clinicians and scientists working on neurological and psychiatric aspects of HIV infection. It

re-emphasises the fascination and productivity of the combined approach of both disciplines.

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NOTICE

The UK Family Planning Association is beginning a research project to explore the development of recent family planning and reproductive health initiatives within the UK. We particularly wish to gather information from people currently or recently associated with clinical facilities which combine access to family planning information and services with genitourinary medicine and sexual health provision. In the first instance we wish to invite health and social welfare professionals with experience of planning, developing, managing or working in combined clinic services to contact:

Joan Walsh, Health Policy and Research Officer, Family Planning Association, 27-35 Mortimer Street, London W1N 7RJ, UK. Telephone: 071 636 7866.

Correction

Higgins *et al*: Breast abscess due to *Pseudomonas aeruginosa* in an HIV antibody positive man (*Genitourin Med* 1994;70: 147-8). The name of the last author was incorrectly spelled and should have been Dr Penny Chandiok.