

was estimated if both high performance testing and pooling are assumed (table 1).

We conclude that with pooling and application of high performance testing major improvements in cost effectiveness of screening women for asymptomatic CT can be obtained.

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Anti-HIV serology in patients with sexual dysphoria in screening test before sex change surgery

The health and behavioural issues of homosexual men and women have recently become a focus of research and interest. A well conceived framework within which to consider the uniqueness of problems faced by homosexual youths and the role of healthcare providers is needed.¹ Significant physical morbidity occurs among homosexual men and women because healthcare providers are often unaware of their actual or potential

Table 1 Demographic data and anti-HIV serology

Demographic	Anti-HIV serology	
	Positive (n=2)	Negative (n=33)
Sex		
Male	2	31
Female	0	2
Occupation		
Beauty salon workers	1	17
"Gay show" workers	1	12
Private business	0	2
Secretary	0	1
Student	0	1
Injecting drug use		
Ever	0	0
Never	0	33
Previous plastic surgery		
Ever	1	5
Never	0	29
Hormone injection		
Ever	0	20
Never	0	15
Abnormal sexual intercourse		
Ever	2	14
Never	0	19

health concerns. Physical health concerns mainly include HIV disease, hepatitis, and other sexually transmitted diseases. Healthcare professionals, who are clinically competent in the care of homosexual men and women, should have the opportunity to reduce the risk of disease, while providing unbiased, quality care which recognises the unique problems of this population.² In this study, we report the prevalence of HIV infection among the homosexual men and women who visited the pre-admission clinic, King Chulalongkorn Memorial Hospital, Bangkok, for further sex change surgery.

A prospective study on the data concerning anti-HIV test for 35 cases (33 homosexual men and two lesbian women) with sexual dysphoria who attended the pre-admission clinic, King Chulalongkorn Memorial Hospital, Bangkok for further sex change surgery, during years 1999 and 2000 was performed. The demographic data about occupation, injecting drug use, previous plastic surgery, hormone injection, and abnormal sexual intercourse (as oral and anal sex) were also reviewed for each case.

For all 35 cases of sexual dysphoria, only two cases of anti-HIV seropositivity were detected. The prevalence was equal to 5.71%. These two cases were homosexual. The demographic data of HIV seronegative and HIV seropositive cases are shown in table 1.

Currently, the two major routes of transmission of HIV are blood borne and sexual propagation. Sexual propagation also includes the abnormal sexual behaviour such as oral and anal sex found in the "gay" population.³ Unique aspects of Thai culture have shaped the response of homosexual men and women to HIV infection in Thailand. Thailand is a relatively homogeneous society that has, by and large, felt invulnerable to AIDS, viewing it primarily as a Western phenomenon. This attitude has also been common in the gay community and has resulted in some homosexual men and women engaging in high risk behaviour.

In Thailand it has been argued that HIV infection is still a major health problem

among homosexual men and women. The current HIV epidemic among young homosexual men and women is a major public health concern. Nevertheless, hardly any specific HIV education interventions have been designed for this population. In this study, the rather high rate of HIV infection among the homosexual men and women attending the hospital for further sex change surgery was detected. Compared with the rate in the general population in Thailand,⁴ this rate is five times higher. Therefore, this population is still a target group for HIV infection, and thus, proper control for this population is necessary.

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Neuropsychiatric reaction induced by clarithromycin

We read with interest the case report by Prime and French¹ describing a person with HIV infection who developed a severe neuropsychiatric reaction during clarithromycin, zidovudine, didanosine, and nevirapine treatment. The authors suggest that this reaction was caused by the clarithromycin and not the antiretrovirals. Indeed, central nervous system (CNS) symptoms are a known side effect of clarithromycin.² CNS adverse effects, however, have also been reported with zidovudine³ and efavirenz.⁴

So far with nevirapine neuropsychiatric side effects have not been described. For this reason we would like to report the case of a patient who developed CNS side effects shortly after starting nevirapine.

A 40 year old woman with HIV infection was initially treated with ritonavir, saquinavir, and stavudine. Because she developed lipodystrophy she was switched to nevirapine, lamivudine, and zidovudine. Shortly after starting this treatment, she started to feel depressed and to experience bad dreams. Her CD4+ lymphocyte count was 727 × 10⁹/l and her viral load was undetectable. She was living under stressful conditions (her husband was also living with HIV) but according to her there was no recent change in her life to explain this depression. The nevirapine was replaced by abacavir and from then on the CNS side effects rapidly disappeared.

This case report strongly suggests that the nevirapine was responsible for the CNS symptoms.

CNS side effects related to antiviral treatment may be caused by high drug levels. Clarithromycin is known to increase nevirapine levels by about 26%.⁵ The fact that in the patient described by Prime and French the neuropsychiatric symptoms disappeared within 72 hours after stopping the clarithromycin suggests this drug was responsible for

causing these symptoms. However, it is also possible that after stopping the clarithromycin the nevirapine levels decreased and that therefore potential nevirapine related side effects disappeared. We propose that in HIV clinical trials patients should be monitored more closely for possible neuropsychiatric side effects and that if these side effects appear antiretroviral drug levels should be measured.

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Immune reconstitution eosinophilia due to schistosomiasis

A 21 year old black African heterosexual woman, formerly resident in south east Africa, presented in early October 2000 on arrival in the United Kingdom. Examination revealed hepatosplenomegaly. Investigation showed Hb = 10.2 g/dl, WBC = $2.9 \times 10^9/l$ (neutrophils = 1.5, lymphocytes = 0.8, and eosinophils = $0.3 \times 10^9/l$). Blood urea and electrolytes were normal. Liver function tests showed alkaline phosphatase = 1704 (normal = 45–122) U/l, and alanine transaminase = 65 (normal = 7–63) U/l. Hepatitis A and C serology was negative; hepatitis B serology showed sAg negative and cAb positive. Serum α fetoprotein was negative. An HIV-1 antibody test was positive, plasma HIV-1 RNA level >75 000 copies/ml, CD4 count = 170 cells $\times 10^9/l$. An abdominal ultrasound confirmed hepatosplenomegaly; there was no intra-abdominal lymphadenopathy and no ascites. A chest radiograph showed micronodular shadowing throughout both lungs. In order to rule out the possibility of tuberculosis, bronchoscopy was performed. Staining and culture of bronchoalveolar lavage fluid was negative for bacteria, mycobacteria, fungi, and parasites. A bone marrow biopsy showed only non-specific reactive changes; culture was negative. A liver biopsy showed portal fibrosis with a moderate chronic inflammatory infiltrate, but no cirrhosis; a schistosomal egg was seen. Schistosomal antibodies were detected by ELISA, positive at level 4 (optical density = 0.767). A diagnosis of schistosomiasis was made.

The patient began highly active antiretroviral therapy (HAART) with stavudine, lamivudine, and efavirenz on 1 December

2000. Four weeks after starting HAART the HIV RNA level had fallen to 25 000 copies/ml, CD4 count remained at 170 cells $\times 10^9/l$ but the eosinophil count had risen to $0.8 \times 10^9/l$ (normal = $0.04\text{--}0.44 \times 10^9/l$). After 4 months of HAART the HIV level was below the limits of detection, the CD4 count had risen to 230 cells $\times 10^9/l$, and the eosinophil count had further risen to $1.5 \times 10^9/l$. At this time the patient agreed to treatment of schistosomiasis with praziquantel (40 mg/kg in two divided doses in 1 day). Following praziquantel the eosinophil count fell to $0.5 \times 10^9/l$.

The development of eosinophilia in this HIV infected patient with schistosomiasis occurred in the context of a falling HIV RNA level and an increase of CD4 count, indicating partial immune reconstitution.

Partial restoration of cell mediated immunity induced by antiretroviral therapy, as demonstrated by recovery of partial CD4+ T lymphocyte reactivation to memory antigens,^{1,2} may cause development of an inflammatory response, in this case eosinophilia, in patients latently affected with opportunistic and non-opportunistic pathogens. Reactivation mycobacterial lymphadenitis,³ cryptococcal meningitis,⁴ and cytomegalovirus retinitis⁵ and pneumonitis⁶ have been described. The case described here suggests that development of an eosinophilia to schistosomiasis should be added to the list of immune reconstitution phenomena.

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Cost effectiveness analysis of a population based screening programme for asymptomatic Chlamydia trachomatis infection in women

With reference to the article by van Valkengoed *et al*,¹ we would like to express our views.

We agree with the authors' statement that systematic screening of all women aged 15–40 years for asymptomatic *Chlamydia trachomatis* infection is not cost effective, especially when the prevalence of infection in Amsterdam is low (2.2%–2.8%). Not all countries have achieved such low levels. Even in England and Wales where the prevalence of the infection is higher it is not cost effective to screen all women. However, computer modelling performed for the chief medical officer's expert advisory group on *Chlamydia trachomatis*² in the United Kingdom and other countries^{3,4} has shown that it is cost effective to screen populations where the prevalence is 3%–6%. The Chlamydia Pilot Study, which was conducted in Wirral and Portsmouth in 1999–2000, detected a prevalence of chlamydial infection of approximately 10% in women aged between 16 and 25.⁵ There is, therefore, a strong argument for screening this age group in the United Kingdom at the present time and not above 25 years as prevalence above this age is low.

One must be careful when extrapolating data from a different country with a different population. However, it would be wise to consider that in the future in the United Kingdom, when screening is established, the prevalence may fall and the cost effectiveness may be reduced.

Although it is not cost effective to screen men, as there are only minor sequelae to be prevented, one shouldn't forget that they are the major reservoir of infection. We should aim not to reinforce existing inequalities by sparing them their share of responsibility for sexual health. Screening men as well will not only decrease the prevalence but also reduce the psychosocial impact of screening for genital chlamydia in women.

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