Sexually transmitted infections

Ten common myths

Antonio AT Chuh
MD, FRACGP, FRCP(Irel), FHKAM(Fam Med), is Associate Professor, Department of Community and Family Medicine, The Chinese University of Hong Kong, Prince of Wales Hospital, Shatin, Hong Kong. achuh@iohk.com

William CW Wong
DCH(UK), MRCGP, is Assistant Professor, Department of Community and Family Medicine, The Chinese University of Hong Kong, Prince of Wales Hospital, Shatin, Hong Kong.

Albert Lee
MD, FRACGP, FFPH(UK), FRCP(Irel), FHKAM(Fam Med), is Professor and Head in Family Medicine, Department of Community and Family Medicine, The Chinese University of Hong Kong, Prince of Wales Hospital, Shatin, Hong Kong.

BACKGROUND
The management of sexually transmitted infections serves as a good example of how medical practitioners should offer continuous and 'whole person' care to patients and their contacts.

OBJECTIVE
This article discusses 10 myths commonly held by patients with sexually transmitted infections consulting their general practitioners.

DISCUSSION
We stress the importance of risk assessment, patient education, pre- and post-test counselling, assessment of associated diseases, contact tracing, and modification of health related behaviour in managing patients with sexually transmitted infections.

Myth 1

There is no difference between HIV and AIDS

The general practitioner cannot assume that the patient understands the difference. Patients requesting a HIV test might state they would like to be tested for AIDS. The lack of understanding of the natural course of the disease also leads to assumptions that people with HIV or AIDS have early signs or symptoms.

The importance of pre- and post-test counselling for HIV testing should not be underestimated (*Table 1*).[1] Counselling should not be neglected whether HIV antibodies are offered as a single test, or as part of a panel of tests in screening for sexually transmitted infections (STIs).

It is controversial as to whether the 'window period' concept should be discussed during pretest counselling. The Centers for the Diseases Control and Prevention currently do not recommend such discussion on the ground that such issues 'could confuse the client and diffuse the importance of the HIV prevention message'.[2] We believe that the patient's educational level and their background knowledge of HIV should be factors to consider in deciding whether the 'window period' concept should be discussed.

Myth 2

Genital herpes is only caused by herpes simplex 2 virus

About 25–30% of genital herpes cases are caused by herpes simplex virus 1 (HSV1). The proportion of genital herpes related to HSV1 is increasing in developed countries.[3] For first episode genital infections there is an increasing belief that HSV1 is the predominant cause, while HSV2 remains the predominant cause of recurrent attacks.[4,5] This may be related to delayed exposure of children and adolescents to oral HSV1 infection, and an increase in the practice of oro-genital contact.[6]

It is likely that genital herpes, whether caused by HSV1 or HSV2, is sexually transmitted.[4] Around 4% of oro-facial herpes and around 40% of herpes from other extragenital sites (hands, fingers, abdomen, feet) are caused by HSV2.[7] It is likely that extragenital herpes, if caused by HSV2, is also sexually transmitted, although the evidence is less solid.

Serology is not the ideal screening or diagnostic test for genital herpes. If serological investigations are ordered, the GP should inform the patient that negative serology result against HSV2 does not exclude genital herpes, as it can be caused by HSV1, or the patient could be in the window period before antibodies to HSV2 develop.

Table 1. HIV pretest counselling

Assess risk
Assess understanding
Provide information
Discuss implications of positive and negative results
Modify risk behaviour
Discuss record keeping
Determine which test(s) to order (eg. serology/serology + PCR)
Determine the optimal time of testing and post-test visit
Obtain and record informed consent
Assess social support
Discuss disclosure of results to sexual partner(s)
Provide support and follow up

Myth 3

With new antiviral therapies, genital herpes is now a curable disease

Like other herpes virus, HSV1 and HSV2 exhibit the pathogenic properties of lifelong latent infection and reactivation. Antiviral therapies have mainly two roles in genital herpes infection: treatment of an acute attack, and as prophylactic therapy. There is however, no evidence that the latent infection can be permanently 'cured' by existing antiviral therapies.[8]

A related myth is: 'If I have sex when I am not experiencing a herpes relapse, I will not infect my partner'. Many patients are unaware of asymptomatic viral shedding. The infectivity is probably lower when the patient is not suffering a clinical relapse. However, during a clinical relapse many patients will refrain from sexual activities. Therefore, most transmissions of genital herpes occur when the 'donating' partner is asymptomatic.[9]

Myth 4

If I have sex when I do not have visible genital warts, I will not infect my partner

Asymptomatic viral shedding also occurs for human papillomavirus (HPV).[10] As in genital herpes, it is likely that many transmissions occur when the 'donating' partner is asymptomatic.

A related myth is: 'If my sex partner does not have symptoms, he/she is not infected with HPV'. The patient with genital warts (*Figure 1*), or a history of genital HPV infection, should be advised that asymptomatic and subclinical infections of HPV are common.[11] It is vitally important that the female partner(s) be examined and have regular Pap tests.[12]

Unfortunately, a sensitive test to diagnose asymptomatic HPV infection in men does not exist. Even polymerase chain reaction of genital skin swabs and urine has been reported to have low sensitivity in men.[13]

Myth 5

Most cases of male urethritis are caused by gonorrhoea and can be cured with a single injection

For male urethritis with an identifiable cause, the most likely culprit is *Chlamydia trachomatis* serovars D–K.[14] The clinical significance is that if the patient does not realise that urethritis is most likely caused by pathogens other than *Neisseria gonorrhoeae*, he may not be compliant with completing the course of oral antibiotics. The use of single dose regimens such as oral azithromycin 1 g is helpful in ensuring patient compliance as well as minimising the development of antibiotic resistance.

The role of *Trachomatis vaginalis*[15] and HSV[16] in male urethritis are probably underestimated. *Mycoplasma spp.* including *Ureaplasma urealyticum*,[17] *M. hominis*,[17] and *M. genitalium*[18] are likely to be aetiological agents for male urethritis. More investigation is necessary to determine their importance in management.

Myth 6

The female equivalent of male urethritis is urinary tract infection

By 'equivalent' we denote diseases caused by similar organisms. The clinical manifestations, investigations, and treatments can be different for both sexes. At the risk of oversimplification, we usually inform our patients that the female equivalent of male urethritis is endocervicitis. For some male patients with balanitis, the female equivalent is vulvovaginitis.

The clinical implication is that the partner(s) of the index patient may have a subclinical or asymptomatic infection. Investigations for the sexual partner may be different. For example, the documentation of male urethritis is usually by the presence of pyuria. For the female partner, a urine specimen may also be collected, but examination of the cervix

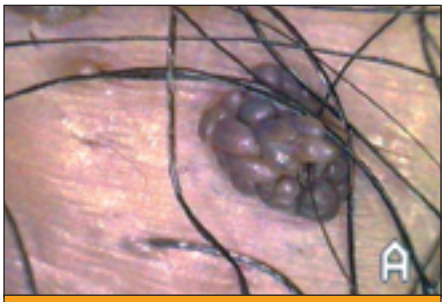

Figure 1. Discrete warty growths on shaft of penis diagnostic of genital warts (condyloma acuminata)

and endocervical swabs are more relevant. Ligase chain reaction testing of urine for *C. trachomatis* (and *N. gonorrhoeae*, if warranted) may be used in asymptomatic individuals to screen for infection with these organisms. For symptomatic individuals, examination with microscopic examination of urethral or cervical discharge may be performed.

Myth 7

Hepatitis B and C are transmissible by sex; hepatitis A isn't

Hepatitis A is transmissible by oro-genital contact.[19] Hepatitis A, B, C, and D are transmissible by sexual activities with hepatitis B, C, and D by both heterosexual and male homosexual routes.[19] Hepatitis C is less commonly transmitted through heterosexual sex, with some reports of transmission via male homosexual sexual activities.

Salmonellosis and typhoid fever are also transmissible by oro-genital contact.[20] Other enteric pathogens such as shigella may also be transmissible by this route.[21] As the major routes of transmission of hepatitis A and salmonella diseases are unrelated to sexual activities, we usually adopt the term 'sexually transmitted infections' in discussion with our patients.

Myth 8

STIs are adult stuff, they do not affect infants and children

Most STIs can incur congenital infections. Sexual abuse can also lead to STIs in children and GPs should be careful not to overlook such a possibility. The onus is on the doctor to discuss the implications of pregnancy and childbirth for patients with a history of genital herpes.[22]

Myth 9

STIs are strictly genital diseases

Virtually all STIs have extragenital manifestations. The clinical significance is that such manifestations are usually regarded by patients as unrelated to their sexual behaviour. We have previously reported a woman in whom a mucous patch in the oral cavity and arthralgia led us to suspect and finally confirm a diagnosis of secondary syphilis.[23] Manifestations of STIs include: the oral cavity, eyes, fingers, joints, cardiovascular system, neurological system, gastrointestinal tract, hepatobiliary system, and the musculoskeletal system. Many of these manifestations are related to HIV infection, complications of common diseases, common diseases in immunocompromised hosts, or congenital infections.

Myth 10

Genital rash or genital mass denotes an STI

Most cutaneous eruptions can affect the genitalia. Conditions such as psoriasis, lichen planus, fixed drug eruption, vitiligo, and lichen simplex chronicus (*Figure 2*) have special predilection for the genital and perigenital skin. Lichen sclerosis et atrophicus for males and females (previously known as balanitis xerotica obliterans for males) are nonsexually transmitted but specific to the genitalia. Common skin problems such as tinea cruris and erythrasma might affect the perigenital skin. Patients and their sexual partners might wrongly attribute such to an STI. Penile papules with a background of severe nocturnal pruritus and generalised excoriation are virtually pathognomonic of scabies (*Figure 3*). These may or may not be sexually transmitted. Pearly penile papules (*Figure 4*) are a normal variant often mistaken by patients to be genital warts.

Figure 2. Lichenification on scrotal skin in lichen simplex chronicus. Chronic pruritus led to a vicious itch-scratch cycle

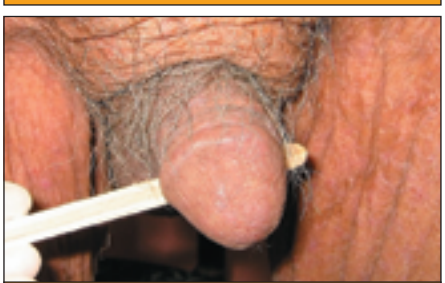

Figure 3. Erythematous penile papules in a patient with noctural pruritus and generalised excoriation. A clinical diagnosis of scabies is obvious. The history is not suggestive of a STI

Figure 4. Rows of small, smooth, and dome shaped pearly penile papules on the corona of penis are normal findings. These papules are angiofibromas or fibropapillomas with no macroscopic feature or microscopic evidence of HPV infection. They are not Tysons glands (secretory glands on either side of frenulum) or Fordyce spots (ectopic submucosal sebaceous glands)

That 'genital rash occurring after sex means STI' is a related myth. There exists evidence that sexual activity can lead to antigen transfer via body fluids such as semen or sweat, and lead to allergic contact dermatitis, urticaria, allergic rhinitis, asthma. We have previously reported that fixed drug eruption can be a sexually inducible reaction (drugs taken by female partners, subsequent rash in male partners),[24] although this eruption is more commonly caused by the direct intake of drugs such as antibiotics or nonsteroidal anti-inflammatory agents.

Conclusion

The dispelling of myths by the GP is important in managing patients with STIs. Management should also include risk assessment, patient education, pre- and post-test counselling, assessment of associated diseases, contact tracing, and modification of health related behaviour.

Conflict of interest: none declared.

References

1. Gallant JE. HIV counseling, testing, and referral. Am Fam Physician 2004;70:295–302.
2. Centers for Disease Control and Prevention. Revised guidelines for HIV counseling, testing, and referral. MMWR Recomm Rep 2001;50:1–57.
3. Tran T, Druce JD, Catton MC, Kelly H, Birch CJ. Changing epidemiology of genital herpes simplex virus infection in Melbourne, Australia, between 1980 and 2003. Sex Transm Infect 2004;80:277–9.
4. Manavi K, McMillan A, Ogilvie M. Herpes simplex virus type 1 remains the principal cause of initial anogenital herpes in Edinburgh, Scotland. Sex Transm Dis 2004;31:322–4.
5. Filen F, Strand A, Allard A, Blomberg J, Herrmann B. Duplex real-time polymerase chain reaction assay for detection and quantification of herpes simplex virus type 1 and herpes simplex virus type 2 in genital and cutaneous lesions. Sex Transm Dis 2004;31:331–6.
6. Malkin JE. Epidemiology of genital herpes simplex virus infection in developed countries. Herpes 2004;11(Suppl 1):2A–23A.
7. Lowhagen GB, Tunback P, Bergstrom T. Proportion of herpes simplex virus (HSV) type 1 and type 2 among genital and extragenital HSV isolates. Acta Derm Venereol 2002;82:118–20.
8. Posavad CM, Huang ML, Barcy S, Koelle DM, Corey L. Long term persistence of herpes simplex virus-specific CD8+ CTL in persons with frequently recurring genital herpes. J Immunol 2000;165:1146–52.
9. Koelle DM, Corey L. Recent progress in herpes simplex virus immunobiology and vaccine research. Clin Microbiol Rev 2003;16:96–113.
10. Broker TR, Jin G, Croom-Rivers A, et al. Viral latency: the papillomavirus model. Dev Biol (Basel) 2001;106:443–53, 465–75.
11. Zanotti KwM, Belinson J. Update on the diagnosis and treatment of human papillomavirus infection. Cleve Clin J Med 2002;69:948, 951–5.
12. Centers for Disease Control and Prevention. Genital HPV infection. Available at www.cdc.gov/std/HPV/STDFact-HPV.htm
13. Fife KH, Coplan PM, Jansen KU, et al. Poor sensitivity of polymerase chain reaction assays of genital skin swabs and urine to detect HPV 6 and 11 DNA in men. Sex Transm Dis 2003;30:246–8.
14. Tait IA, Hart CA. Chlamydia trachomatis in non-gonococcal urethritis patients and their heterosexual partners: routine testing by polymerase chain reaction. Sex Transm Infect 2002;78:286-8.
15. Schwebke JR, Hook EW 3rd. High rates of Trichomonas vaginalis among men attending a sexually transmitted infections clinic: implications for screening and urethritis management. J Infect Dis 2003;188:465–8.
16. Lautenschlager S, Eichmann A. Urethritis: an underestimated clinical variant of genital herpes in men? J Am Acad Dermatol 2002;46:307–8.
17. Kilic D, Basar MM, Kaygusuz S, Yilmaz E, Basar H, Batislam E. Prevalence and treatment of Chlamydia trachomatis, Ureaplasma urealyticum, and Mycoplasma hominis in patients with non-gonococcal urethritis. Jpn J Infect Dis 2004;57:17–20.
18. Uuskula A, Kohl PK. Genital mycoplasmas, including Mycoplasma genitalium, as sexually transmitted agents. Int J STD AIDS 2002;13:79–85.
19. Brook MG. Sexually acquired hepatitis. Sex Transm Infect 2002;78:235–40.
20. Reller ME, Olsen SJ, Kressel AB, et al. Sexual transmission of typhoid fever: a multistate outbreak among men who have sex with men. Clin Infect Dis 2003;37:141–4.
21. Marcus U, Zucs P, Bremer V, et al. Shigellosis – a re-emerging sexually transmitted infection: outbreak in men having sex with men in Berlin. Int J STD AIDS 2004;15:533–7.
22. Brown ZA, Wald A, Morrow RA, et al. Effect of serologic status and caesarean delivery on transmission rates of herpes simplex virus from mother to infant. JAMA 2003;289:203–9.
23. Zawar V, Chuh A. Mucous patches and arthralgia: a presentation of secondary syphilis not to be missed. J R Soc Med 2004;97:79–80.
24. Zawar V, Kirloskar M, Chuh A. Fixed drug eruption: a sexually inducible reaction? Int J STD & AIDS 2004;15:560–3.

afp CORRESPONDENCE email: afp@racgp.org.au