

PostScript

LETTERS

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HIV epidemicity in context of STI declines: a telling discordance

Decosas and Padian report, but do not discuss, a noteworthy disassociation in epidemic trajectory between human immunodeficiency virus (HIV) and sexually transmitted infections (STI) in Zimbabwe.¹ They cite estimates that, between 1990 and 1999, HIV prevalence increased linearly from 9% to 25%, while STI syndrome reports declined substantially, from 963 436 cases to 727 788. The authors not only believe that observed STI declines are real, but cite increases in reported condom use by high risk people (for example, prostitute women, truck drivers, miners, and young people) as supporting evidence. What is not clear is why HIV prevalence would increase markedly coevally with increasing condom use in high risk populations and with decreasing STI incidence. Assuming synergism between STI and HIV transmission,² one would expect that a burgeoning and sexually mediated HIV epidemic would be accompanied by corresponding increases in STI transmissions. An estimated increase in HIV prevalence from 9% to 25% in a decade, implying a 12% annual epidemic growth rate, is not likely to be due to differences between HIV, a chronic infection that accumulates in a reservoir, and STI, which tend not to. Does this anomaly require clarification?

Recent analyses^{3,4} suggest that a large proportion of HIV infections, especially in sub-Saharan Africa,³ may be a consequence of unsafe medical injections. This unsuspected and scientifically underexplored transmission vector is overlooked by the authors as well (exception: "blood safety" in fig 1). Theirs is not the first report of an epidemiologically suspicious anomaly between STI and HIV trends in Africa⁶ and, if others' suspicions are correct,^{3,5} it is unlikely to be the last.

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Vaginal infection by *Enterobacter sakazakii*

In August 2001, a 26 year old woman seen at our outpatient clinic in Budapest complained of vulvar pruritus and vaginal discharge in the preceding 2 weeks. The only risk factor she admitted was that she had bathed in the resort lake Balaton a few times a week before the onset of symptoms, when the water was unusually warm (26-28°C). Examination revealed vulvovaginitis with mucous discharge at pH 5.5. A vaginal smear showed a large number of polymorphonuclear leucocytes, Gram negative rods, but no *Lactobacillus*. Culture on blood agar at 37°C for 48 hours resulted in yellow pigmented, bright, tough colonies. Biochemical analysis¹ verified *Enterobacter sakazakii*. Standard disc diffusion technique,² on Mueller-Hinton agar (Becton Dickinson, Sparks, MD, USA) using commercial discs (Oxoid, Basingstoke, UK), revealed sensitivity to carbenicillin, netilmycin, cefazolin, ofloxacin, tetracycline, and gentamicin; limited sensitivity to erythromycin; and resistance to ampicillin, clindamycin, nalidixic acid, furadantin. This pattern is common in recent isolates.³ Empirical treatment—starting before laboratory data were available—with intravaginal Pimafucort ointment (1% natamycin, 0.25% neomycin, 0.5% hydrocortisone) for 7 days resulted in a complete recovery in 2 weeks, at which time the vagina was colonised by *Streptococcus agalactiae*. The following week a normal *Lactobacillus* flora at pH 4.2 returned.

Nearly 50 *E sakazakii* infections resulting in meningitis, necrotising enterocolitis, and one urine infection of newborns have been documented. *E sakazakii* have been recovered from

their blood, spinal fluid, throat, trachea, stomach aspirates, and rectum. Newborns were premature, and fed on powdered milk formula, which is the known source of infection.^{3,4} The means of its contamination is not known. For affected newborns, vaginal delivery does not seem to be a risk factor, since no colonisation of the mother's genital tract has been reported. Less than 20 isolates have been obtained from children and adults, including eight from urine and one from genital secretion. Patients had either neonplasm or other serious underlying conditions, but their clinical details were not available. In both newborns and adults occasional coinfections with *Serratia marcescens*, *Staphylococcus aureus*, *Enterococcus faecalis*, or *Candida albicans* were reported.^{3,5}

The normal habitat, reservoir, and pathogenesis of *E sakazakii* are unknown. No cultivation from environmental sources (surface water, animals, raw cow's milk, etc) has been successful,³ except recently from the gut of fruit flies.⁶ The rare related species *E intermedium* causing urinary tract infection, was found in drinking water and soil.³ Our case strongly suggested that warm surface water was the source of infection. The unusually high thermal resistance of *E sakazakii*³ might contribute to its survival during the manufacture powdered milk batches.⁴ It is important to remember that the stomach of newborns, especially that of premature babies, lacks free acid and is less acidic than that of adults. Ingested milk preparations remaining neutral or slightly alkaline in their stomach ensure survival and subsequent infection in the alkaline intestine. This pathomechanism is similar to pH increase in vaginal infections in the lack of *Lactobacillus* flora. The detrimental effect of *E sakazakii* infection is also reflected by the fact that recolonisation by the normal flora and a pH shift towards physiological level could be achieved only gradually in our patient.

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Successful use of valaciclovir in a case of recurrent urticaria associated with genital herpes

Urticaria is a common skin condition but the symptoms and signs can be extremely distressing. The condition is often idiopathic.¹ The management of urticaria can be frustrating even when triggers are identified.² We describe a case of recurrent urticaria associated with genital herpes attacks and a successful use of suppressive therapy with valaciclovir.

A 35 year old white woman presented in March 2000 to a genitourinary medicine (GUM) clinic with 5 year history of recurrent lesions typical of genital herpes simplex virus (HSV) infection occurring almost every month. She had also been developing physical urticaria manifesting as itchy wheals on pressure areas of the body, approximately 24 hours before the onset of genital HSV attacks. The lesions showed no characteristics of erythema multiforme or vasculitis. Each urticarial attack lasted from 20–30 minutes and had a cholinergic element being exacerbated by exercise and heat. Antihistamines were not effective. She had contact sensitivity to perfumes, make up, and coloured bath products. There was no history of angioedema, other atopic disease, or drug allergies. She was otherwise well and on no medication.

Culture for HSV was negative. However, serology was positive for HSV type 1 IgG antibody. HSV type 2, hepatitis B and C serology were negative. Her IgE level was normal. Immunological investigations including CD4/CD8 count showed no evidence of immunodeficiency.

Her HSV attacks were frequent and distressing. She began suppressive therapy with valaciclovir 500 mg twice a day and antihistamines in March 2000 and the symptoms were well controlled. Both urticaria and herpes recurred when the valaciclovir therapy was discontinued after 6 months. She recommenced her therapy in October 2000 but required higher doses of valaciclovir to control her symptoms. She is currently taking valaciclovir 1 g twice daily and cetirizine 10 mg daily. She had a single episode of urticaria associated with genital herpes precipitated by intense sunlight exposure in September 2001 and had no further attacks since then.

Urticaria can be triggered by a large number of diverse allergens including sexually transmitted infections such as hepatitis B virus.³ A case of chronic urticaria preceding genital herpes and a successful treatment with a short course of aciclovir has been described. However, the long term follow up of this patient was not known.⁴ A recent study in Ohio has reported a successful use of maintenance therapy with aciclovir in five out of 12 patients with chronic urticaria; none had genital herpes.⁵ In our patient, control of both genital herpes and recurrent urticaria with valaciclovir therapy suggests a close temporal

association between the onset of urticaria and development of herpetic lesions. The exact role of HSV in the pathogenesis of urticaria is unknown but it may be related to hypersensitivity reaction to viral antigens. Antiviral agents may be effective by suppressing these antigens.

This case demonstrates the importance of early recognition of urticaria associated with HSV infections in order to avoid delay in instituting antiviral treatment in GUM clinics or other settings for this disabling skin condition.

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Inhibition of *Neisseria gonorrhoeae* by vaginal lubricants

Microbiology culture remains the diagnostic standard for gonococcal infection. Isolation of the pathogen confirms the clinical diagnosis and allows assessment of the antimicrobial susceptibility of the gonococcal strain, guiding effective therapy. The sensitivity of endocervical swab culture has been reported as 80–90% but this varies with the quality of culture media and adequacy of the specimen.¹ The gonococcus is a nutritionally demanding bacterium, readily inhibited by adverse culture conditions.

For decades, doctors in genitourinary medicine have been advised to lubricate metal vaginal specula with water only. With the increasing use of disposable vaginal specula, which are more difficult to insert, some workers have promoted the use of vaginal lubricants, such as KY jelly, to reduce patient discomfort during clinical examination and specimen collection.



Figure 1 Five randomly selected clinical strains of *Neisseria gonorrhoeae* growing on gonococcal sensitivity agar. Note the central region of the plate where a line of KY jelly has inhibited growth.

Figure 1 shows five randomly selected clinical strains of *Neisseria gonorrhoeae* inoculated by swab (10⁶/ml) and growing readily on gonococcal sensitivity agar except in the central region of the plate where a line of KY jelly has inhibited growth.

Some studies have demonstrated the inhibitory actions of various vaginal lubricants, including KY jelly, against *N gonorrhoeae*,² while others have found KY jelly to be relatively non-toxic to both chlamydia and gonococci.³

In view of these conflicting findings, prospective studies are required to assess the clinical significance of using vaginal lubricants when collecting specimens for gonococcal culture. Pending the completion of such studies we recommend that vaginal lubricants should not be used when obtaining endocervical samples for microbiological investigation.

Contributors

REH, design of practical work, literature review, production of first draft of manuscript; JDJ, performance of practical work, literature review, critical comment on draft manuscript; FD identification of clinical issue, literature review, critical comment on draft manuscript.

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