LETTERS TO THE EDITOR

Carbamazepine in Reiter’s syndrome

Editor,—A psoriatic spectrum with Reiter’s syndrome as the most severe manifestation occurs with greater frequency in HIV infected individuals. Immunosuppressive therapies for BS are associated with a poor response and increased morbidity.1 We describe a case where carbamazepine showed an excellent response in an HIV infected patient with Reiter’s syndrome.

A 30 year old married man presented with erythematous papules and plaques of 2 months’ duration covered with hard limpet-like scales on face, body, and both extremities (fig 1). Pains and trophic lesions showed keratoderma blennorrhagicum and subungal hyperkeratosis with distal onycholysis. Both knees and wrists had painful swelling with restriction of movements. With this clinical presentation Reiter’s syndrome was inferred. All routine investigations were normal except a raised erythrocyte sedimentation rate of 100 mm in the first hour. x Rays of the affected joints were normal. ELISA for HIV-1 and HIV-2 was positive with two kits (Immunocomb, Tri-dot) and confirmed with western blotting technique (Speciality Ranbaxy Limited). The absolute helper T lymphocyte count was 435 cells/l06. Human leucocyte antigen B27 and rheumatoid factor were negative. The patient was commenced on prednisolone by mouth 60 mg daily and indomethacin by mouth 25 mg three times daily without any concomitant antiretroviral therapy. New erythematous papules and plaques appeared with no relief in joint pain and swelling.

In seeking an effective treatment, we serendipitously came across the efficacy of carbamazepine in an HIV infected patient with psoriatic erythroderma.2 We started carbamazepine 200 mg daily in two divided doses in addition to above. The erythema cleared rapidly within 7 days. To confirm the effect of carbamazepine, it was stopped. New lesions similar to the old ones appeared within 3–4 days. Carbamazepine was then reintroduced in the same dose. Erythema cleared again within 7 days followed by scaling and joint swelling and pain. New lesions stopped appearing. Prednisolone was then tapered off rapidly and analgesics were stopped. Carbamazepine was continued in the same dose for 6 months. On follow up at 1 year, the patient showed no recurrence of skin lesions and synovitis, no change in liver and renal function tests, with no further deterioration in his overall health and no opportunistic infections.

It has been proposed that in genetically predisposed people, the release of neuropeptides like substance P, calcitonin gene related peptide, vasoactive intestinal peptide, and calcitonin gene related peptide, vasoactive intestinal peptide, and the inflammatory leucotriene B4 from cutaneous sensory nerves causes local inflammatory responses that trigger psoriasis.3 Stimulated mast cells secrete a number of proinflammatory cytokines and proteases that act similarly.1,4 Carbamazepine significantly inhibits the uptake of sodium ions and calcium ions, and blocks a cyclic AMP mediated calcium influx that is associated with neuropeptide release and control of a slow potassium current.5

The rapid clearing of erythema, secondary to raised levels of neuropeptides, with carbamazepine may have been mediated through inhibition of these neuropeptides and by inhibition of uptake of noradrenaline. The exacerbation and subsequent resolution of lesions on withdrawal and reinstitution of carbamazepine respectively proves its efficacy in our patient. Also, the clinical remission maintained for 1 year after stopping carbamazepine confirms its therapeutic efficacy in Reiter’s syndrome. The therapeutic response seen in our patient conforms to that seen in the HIV-1 positive patient of Smith et al.1

This apparent success adds carbamazepine to the armamentarium against Reiter’s syndrome in an HIV infected patient. This is the first reported case and an evaluation of longer term carbamazepine therapy is warranted.

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Condoms and warts

Editor,—Wen et al1 should be applauded for their attempt to address the key question of whether or not condoms protect people from genital warts. However, some of the major study variables need clarification, as they did not match up with my knowledge of the Sydney Sexual Health Centre (SSHC) database.

The article discussed the issue of “acquisition of genital warts” and was presented as an incidence study. Cases were defined as: “All patients with a new diagnosis of macroscopic genital warts who attended SSHC [in 1996].” However, many of these patients had been previously diagnosed with genital warts elsewhere while others had recurrent lesions. In Australia, most genital warts are managed by general practitioners.2 Conversely, the experience of specialist services is biased towards recurrent and difficult cases. “New diagnosis” in this situation means new to the clinic but not necessarily new to the patient. This means that the main outcome measure was a mixture of incident, prevalent, and recurrent cases, with the possibility that the warts may have affected the behaviour of many of the study subjects.

The SSHC database does document whether a person has previously been diagnosed with HPV infection. To me, the study would have had more validity if patients with a past history had been excluded.

The diagnostic grouping for warts at SSHC does not distinguish between genital and anal lesions. The readers of the journal need to know that many of these male “genital wart” cases would have been homosexually active men with anal warts. This is important as risk factors for penile and anal warts may differ, potentially confusing the results of the present study.

Originally developed as an HIV risk measure, the condom use variable at SSHC only refers to the previous 3 months or since the last registration/disease episode. Wen et al1’s article failed to mention that this variable was time limited. As 3 months is the median duration before the appearance of exophytic warts,1 up to half of the relevant sexual behaviour may have been overlooked. Finally, the referent group in the table describing condom use deemed as “Not applicable, no sex” should have been more accurately described as “No vaginal or anal sex in the previous 3 months.” Many of these people would have practised oral sex or other sexual acts during those 3 months. Others may have ceased practising vaginal or anal intercourse up to 3 months earlier because of their persistent or recurrent warts.

Large relational quality assured clinical databases can be powerful tools for health service evaluation, surveillance, and the generation of research questions. It may be prudent for researchers to engage the populaation at risk responsible for designing and maintaining those databases to minimise errors of interpretation.

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Reply

Editor,—We are grateful to Dr Dayan for her helpful and constructive comments. The major criticism of our paper relates to the selection of cases, and the possible inclusion
of prevalent and recurrent cases as well incident cases. However, our concern with this possible bias at the outset of the study led us to exclude all patients with a history of previous genital warts. This included those previously diagnosed at SSHC, and those who gave a history of having their warts managed elsewhere. Consequently, when we state a new diagnosis of genital warts, this is precisely what we mean.

With regard to the conduct of the study, this was performed with the assistance of the current data manager responsible for the SSHC data base, whose help and assistance were duly acknowledged.

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12 MINDEL
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Photosensitivity reaction to efavirenz

EDITOR,—The non-nucleoside reverse transcriptase inhibitor (NNRTI) efavirenz is a recent addition to the armamentarium available to physicians in the treatment of HIV infection. However, at present the known side effect profile of this new agent is still in its infancy. We would like to report a case of photosensitivity associated with efavirenz.

A 27 year old white homosexual man was commenced on combivir (zidovudine/ lamivudine) and efavirenz in March of 1999. One month later he reported that he was well and had no major side effects associated with his new combination. However, 4 weeks further into treatment he represented with an itchy rash affecting his arms and hands. On examination there was a maculopapular rash over the affected area but there was no oral ulceration, conjunctivitis, or fever. A drug reaction was diagnosed and he was prescribed antihistamines and asked to continue his medication. One week later the rash had settled, he then commenced combivir and efavirenz.

Photosensitivity in the context of HIV has been reported as a presenting sign of underlying HIV infection in a number of cases.1 In addition to this porphyria cutanea tarda (PCT) has been reported in the context of HIV infection and has been associated with concomitant hepatitis C infection; however, screening for both these conditions was negative. Switching from nevirapine to efavirenz in this context may have been regarded as unwise; however, of 19 patients who have been intolerant of nevirapine secondary to the development of rash, who have switched to efavirenz only nine have developed a mild to moderate rash, of which only two needed to discontinue therapy.1 Photosensitivity in the context of HIV infection may not only be a presenting condition but also secondary to concomitant treatment.

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Accepted for publication 20 March 2000

HIV associated cytomegalovirus retinitis in Melbourne, Australia

EDITOR,—We report the results of a 12 year review of human immunodeficiency virus (HIV) associated cytomegalovirus (CMV) retinitis in Melbourne, Australia.

We conducted a retrospective review of all HIV infected patients diagnosed with CMV retinitis at Fairfield Hospital and the Alfred Hospital between 1984 and 1996, aiming to identify factors at diagnosis of CMV retinitis which were predictive of outcome. Both hospitals had the same protocol for the treatment of CMV retinitis and employed 3 monthly ophthalmological screening of all HIV infected patients with CD4 counts of less than 50 x10^4.

The study outcomes were visual loss and death. Moderate visual loss was defined as visual acuity of less than 6/12 in the better eye, and severe visual loss as visual acuity of less than 6/60 in the better eye (this is legal blindness in Australia).

CMV retinitis was diagnosed in 212 of 1281 patients (16.5%) with AIDS over the study period. As of June 1998, 193 (93%) had died, at a median time of 36 weeks (range 0–192) from CMV diagnosis. Seventy four patients (35%) developed moderate visual loss at a median time of 23 weeks (range 0–163) and 30 patients (14%) developed severe visual loss at a median time of 35 weeks (range 0–120) from diagnosis of CMV retinitis.

The presence of visual symptoms at diagnosis of CMV retinitis was predictive of the development of moderate visual loss (relative risk 2.1, 95% confidence interval 1.4–2.2). Fifty eight of 138 patients (42%) with visual symptoms at diagnosis developed moderate visual loss, compared with 16 of 64 patients (25%) who were asymptomatic at diagnosis (p=0.02). The presence of visual symptoms at diagnosis was not predictive of the development of severe visual loss, or early death (p>0.2). Other factors measured at diagnosis of CMV retinitis included the patients’ age, CD4 count, weight, visual acuity, and the presence of any previous AIDS defining condition. None of these was associated with the development of visual loss or early death (p>0.1).

The advent of highly active antiretroviral therapy (HAART) has resulted in a reduction in the incidence of new diagnoses of opportunistic infections. Prolonged survival times with CMV retinitis have been demonstrated in patients who achieve immunological recovery with HAART.23 The ability to predict those patients who are at highest risk of visual loss may assist in advising those who may reasonably cease maintenance therapy for CMV retinitis following immune restoration. An understanding of the natural history of CMV retinitis in the pre-HAART years remains important in managing patients who are failing HIV therapy.

The only factor measurable at diagnosis of CMV retinitis that was predictive of outcome was the presence of visual symptoms. The use of routine ophthalmological screening in HIV infected individuals with low CD4 counts aims to detect CMV retinitis before visual symptoms occur. It is possible that visual loss may be prevented by detecting disease before retinal damage occurs. A prospective evaluation is needed to confirm this finding.

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Accepted for publication 20 April 2000
Azithromycin v oxytetracycline for the treatment of non-specific urethritis

EDITOR,—Single dose azithromycin 1 g rather than multidose tetracyclines or erythromycin over several days for the treatment of chlamydial urethritis is becoming more widespread as patient acceptability and improved compliance outweigh cost considerations. However, in men, treatment is often initiated on the basis of discrepant evidence of urethritis before the chlamydial result is available. Relatively few studies report the efficacy of azithromycin in the treatment of non-gonococcal non-chlamydial urethritis (NGNU), but recently published evidence-based guidelines for the management of NSU recommend either doxycycline 100 mg twice daily for 7 days or azithromycin 1 g immediately.1

In this genitourinary medicine clinic azithromycin became first line treatment for all proved or suspected chlamydial infections from 1 April 1998. This retrospective study assessed the efficacy of azithromycin for the treatment of NSU compared with oxytetracycline 250 mg four times daily for 7 days, the previous first line treatment regimen for men with microscopic urethritis in whom no Gram negative diplococci were evident.2

NSU was defined as the presence of at least five polymorphonuclear leukocytes (PMNL) in five or more fields on microscopy of a urethral smear, positive culture of Neisseria gonorrhoea after direct plating onto modified New York culture medium and negative chlamydial screen on ELISA testing (Stya) of a urethral swab. ‘Cure’ was defined as either resolution of symptoms or clearing of previously positive two glass urine. A repeat urethral smear was not examined routinely.

‘Treatment failure’ was defined as persistent PMNL on microscopy of a urethral smear taken because of ongoing symptoms or persistent positive two glass urine test, with the possibility of reinfection denied.

The results (see table 1) demonstrate that azithromycin is as effective as oxytetracycline in curing NSU, and produces fewer follow up occurrences in the asymptomatic patients, but was less evident in the azithromycin treated group, who had a lower default rate overall, as previously reported.3

In conclusion, although the numbers are small, it would appear that azithromycin is an effective treatment for NSU, and can be given at the time of clinical diagnosis, pending the chlamydial result. Financial considerations preclude the use of azithromycin as first line treatment for NSU in many centres, but better compliance resulting in fewer treatment failures, and fewer wasted appointments from defaults may counter the economic argument.4

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<table>
<thead>
<tr>
<th>STIs</th>
<th>NSU</th>
<th>Latent syphilis</th>
<th>Genital herpes</th>
<th>Genital warts</th>
<th>Gonorrhoea</th>
<th>HPV</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>7</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>11</td>
<td>12</td>
</tr>
</tbody>
</table>

Many elderly people maintain heterosexual and homosexual activity. Therefore this age group is at a risk of all sexually transmitted infections.5 In our study, a smaller percentage of older attendees had STIs compared with previous studies.1 4 However, the number of older patients who attended for non-STI management is comparable. The delay between symptom recognition and healthcare presentation is a feature of STI related illness behaviour. The delay behaviour among individuals with suspected STIs is age specific, with longer latency periods experienced by people over the age of 50.1 This finding was seen in our study as well.

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Characteristics of older patients attending genitourinary medicine clinics. Health Care in Later Life 1996;252-7.4

Correspondence to: Dr Nelson David

| Delay in symptom presentation among a sample of older GU clinic attenders. Int J STD AIDS 1999;10:43-6. Accepted for publication 30 April 2000

Tertiary syphilis

EDITOR,—I read Dr Reed’s letter on tertiary syphilis1 with interest.

The regimen he describes for the treatment of early syphilis—arsenic, bismuth, and rhus in the clock aqueous penicillin—was used in our hospital from 1946–8 although daily penicillin in beeswax was also used. It was unclear how much inactive penicillin K was in the commercial product used. The penicillin then used here was higher than in Lincoln (40 000–75 000 units 3–4 hourly). There were 10 treatment failures (reinfections) out of 275 patients described.2

Treponema pallidum remains viable in the CSF even after adequate clinical treatment3 4

Table 1 Comparative age, symptoms, and response to treatment of the two groups

<table>
<thead>
<tr>
<th>1997</th>
<th>1998</th>
</tr>
</thead>
<tbody>
<tr>
<td>Azithromycin</td>
<td>Oxytetracycline</td>
</tr>
<tr>
<td>Number treated</td>
<td>76</td>
</tr>
<tr>
<td>Median age (range)</td>
<td>28 (18–63)</td>
</tr>
<tr>
<td>No with symptoms (%)</td>
<td>35 (46)</td>
</tr>
<tr>
<td>No cured (%)</td>
<td>29 (38)</td>
</tr>
<tr>
<td>No treatment failures (%)</td>
<td>6 (8)</td>
</tr>
<tr>
<td>Outcome uncertain*</td>
<td>41 (54)</td>
</tr>
<tr>
<td>Symptomatic dna</td>
<td>8/35 (23)</td>
</tr>
<tr>
<td>Asymptomatic dna</td>
<td>13/41 (32)</td>
</tr>
</tbody>
</table>

*Originally asymptomatic with clear two glass urine; did not reattend (dna); possibly reinfected.

The results of the two glass urine test did not differ significantly between the two groups but overall was positive in 70% of symptomatic patients compared with only 47% asymptomatic (p<0.01). Its low sensitivity and specificity are likely to be even lower in asymptomatic patients. Default from follow up occurred more frequently in the asymptomatic patients, but was less evident in the azithromycin treated group, who had a lower default rate overall, as previously reported.3
I was delighted when the editor sent me this book and asked me to review it. I had looked forward with anticipation to the original series that were published in the BMJ. I had thought then that each article was just superb and now they are all neatly packed together in this ABC, I am of the opinion that this is an excellent book which achieves its aim completely. On the cover, it says “it is an ideal reference for doctors, nurses and patients and all those not involved in the area of sexual health,” and Professor Adler adds in the foreword that this book will put the profession in touch with the real world, real people, with real problems, and fill a large gap in our knowledge.

John Tomlinson, the editor, has pulled together an excellent group of experts who have practical experience in the field and have managed to condense that experience into a series of short articles, all of which make informative, yet entertaining reading. In my opinion, no specific background is required to gain information from these articles and I have recommended specific sections of this book for individual patients who need to read about their problem.

Those of us who work in sexual medicine were amused that the BMJ had to carry a warning about the sexually explicit material inside and, indeed, John Tomlinson refers to this in the preface and admits that a very small number of readers were offended. However, given the general reticence in society about sexual matters, this is not surprising.

Sexual health is an essential part of having a happy and fulfilling life, and everyone who works in a caring profession should be comfortable when the conversation drifts into areas of sexuality. Patients, who often broach the topic with trepidation, need to be assured of a sensitive hearing. In my opinion, this excellent book will give anyone in the caring profession a good grounding in sexual matters, so that they can explore these areas with patients when appropriate, without embarrassment and have some idea of likely strategies of management.

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Australasian Sexual Health Conference
Ven Troppo, Carlton Hotel, Darwin, Northern Territory, 21–24 June 2000
Further details: Shirley Corley, Conference manager, Dart Associates, PO Box 781, Lane Cove, 2066 NSW, Australia (tel: 02 9418 9396/97; fax: 02 9418 9398; email: dartcon@mpx.com.au).

Imperial College School of Medicine, Division of Paediatrics, Obstetrics, and Gynaecology, Caring for Sexuality in Health and Illness (for healthcare professionals and nurses), jointly with Association of Psychosexual Nursing 27 June 2000
Further details: Symposium Office, Imperial College School of Medicine, Queen Charlotte's and Chelsea Hospital, Goldhawk Road, London W6 0XG (tel: 020 8383 3904; fax: 020 8383 8555; email: sympreg@ac.ac.uk).
Sexual Health and HIV Conference: Facing the Millennium, Portsmouth Marriott Hotel, Portsmouth, 28 June 2000

Further details: Rebecca Mitchell (tel: 023 9286 6796; fax: 023 9286 6769).

6th ESC Congress on Contraception in the Third Millennium: a (R)Evolution in Reproductive and Sexual Health, Ljubljana, Slovenia, 28 June–1 July 2000

Further details: Orga-Med Congress Office, Mr Peter Erazd, Essenestraat 77, B-1740 Ternat, Belgium (tel: +32 2 582 08 52; fax: +32 2 582 55 15; email: orgamed@village.uunet.be).

Imperial College School of Medicine, Division of Paediatrics, Obstetrics, and Gynaecology, Bereavement, 5 July 2000

Further details: Symposium Office, Imperial College School of Medicine, Queen Charlotte’s and Chelsea Hospital, Goldhawk Road, London W6 0XG (tel: 020 8383 3904; fax: 020 8383 8555; email: sympreg@ic.ac.uk).

Imperial College School of Medicine, Division of Paediatrics, Obstetrics, and Gynaecology, New Horizons in Recurrent Pregnancy Loss, 29 June–1 July 2000

Further details: Symposium Office, Imperial College School of Medicine, Queen Charlotte’s and Chelsea Hospital, Goldhawk Road, London W6 0XG (tel: 020 8383 3904; fax: 020 8383 8555; email: sympreg@ic.ac.uk).

XIII International AIDS Conference, 9–14 July 2000, Durban, South Africa

Further details: Congrex Sweden AB, PO Box 5619, Linnegatan 89A, 114 86 Stockholm, Sweden (tel: +46 8 459 6600; fax: +46 8 601 91 25; email: aids2000@congrex.se).


Ethical Issues in International Health Research, Durban, South Africa, 16–21 July 2000 (immediately following XIII International AIDS Conference)

Further details: Marie-Christine Ryckaert, Program director, Ethical Issues in International Health Research, Harvard University, John F Kennedy School of Government, Cambridge, MA 02138, USA (tel: (617) 496-0484 ext 7474; fax: (617) 495-3090; email: Marie-Christine_Ryckaert@harvard.edu).


Further details: PACIFICOF, SA, E Granados, 44, 08008 Barcelona, Spain (tel: +34 93.454.54.00; fax: +34 93.451.74.38; email: gp@pacifico-meetings.com).

MSSVD Clinical Developments Fund

The MSSVD Clinical Developments Fund is asking for applications for funding to support projects that advance the understanding and practice of genitourinary medicine. An amount of £10 000 is available to one or more successful applicant(s). Closing date for application is 25 August 2000. Further details: Dr Keith Radcliffe, Honorary Assistant Secretary MSSVD, Whitall Street Clinic, Whitall Street, Birmingham B4 6DH (tel: 0121 237 5719; fax: 0121 237 5729; email: keith.radcliffe@bscht.wmids.nhs.uk).

3rd Congress of the Baltic Association of Dermatovenerology, 7–9 September 2000, Riga, Latvia

Further details: Professor Andris Y Rubins, Department of Dermatovenerology, Medical Academy of Latvia, K Valdemara Street, 76–75, Riga, LV-1013, Latvia (tel: +(371) 7370395; fax: +(371) 7361615; email: arubins@apollo.lv).

National NCCG Update Meeting, Bromsgrove Stakis Hotel, 23–24 September 2000

Further details: Kathy Taylor (tel: 01384 235207; email: palmtraining@tesco.net).

11th Regional Meeting of International Union against Sexually Transmitted Infections, South East Asian and Western Pacific Branch and 24th National Conference of Indian Association for the Study of Sexually Transmitted Diseases and AIDS, 13–15 October 2000, Chandigarh, India

Further details: Dr Bhushan Kumar, Organising Secretary, 11th Regional Meeting of IUSSSI–Asia Pacific (SE Asia and W Pacific Branch), Department of Dermatology, Venereology and Leprosy, PGIMER, Chandigarh - 160 012, India (tel: +91 (0172) 745530; fax: +91 (0172) 744401/745078; email: kumurbhushan@hotmail.com).

Consortium of Thai Training Institutes for STDs and AIDS—10th STD/AIDS diploma course, Bangkok Hospital, Bangkok (30 Oct–12 Nov) and Prince of Songkla University, Hat Yai, Thailand (13–23 Nov) 30 October–23 November 2000

Further details: Hat Yai Secretariat, Dr Verapol Chandeying, Dept of OB-GYN, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkla 90110, Thailand (fax: (66-74) 446 361; email: cvverapol@ratree.psu.ac.th or Bangkok Secretariat, Dr Thantit Palanuvej, Bangkok Hospital, 189 Sathorn Road, Bangkok 10120, Thailand (fax: (66-2) 286 3013; email: pthanit@email.ksc.net).

Consortium of Thai Training Institutes for STDs and AIDS—International Reunion and Refresher Course on Sexual Health, Lee Garden Plaza Hotel, Hat Yai, Thailand 24–26 November 2000

Further details: Hat Yai Secretariat, Dr Verapol Chandeying, Dept of OB-GYN, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkla 90110, Thailand (fax: (66-74) 446 361; email: cvverapol@ratree.psu.ac.th or Bangkok Secretariat, Dr Thantit Palanuvej, Bangkok Hospital, 189 Sathorn Road, Bangkok 10120, Thailand (fax: (66-2) 286 3013; email: pthanit@email.ksc.net).

CORRECTION

CURRENT PUBLICATIONS

Selected titles from recent reports published worldwide are arranged in the following sections:

Gonorrhoea
Chlamydia
Candidiasis
Bacterial vaginosis
Trichomoniasis
Pelvic inflammatory disease
Syphilis and other treponematoses
Hepatitis
HIV
Human papillomavirus infection
Gonorrhoea
Miscellaneous
Chlamydia

GM Li, Q Chen, SC Wang. Sex Transm Dis 2000;27:115–8

Effects of the immunoglobulin A1 protease on Neisseria gonorrhoeae trafficking across polarized T84 epithelial monolayers.

Charged tmRNA but not tmRNA-mediated proteolysis is essential for Neisseria gonorrhoeae viability.
CH Huang, WC Wolfgang, J Withey et al. EMBO J 2000;19:1098–1107

Chlamydia

Acute primary Chlamydia trachomatis infection in male adolescents after their first sexual contact.

Evaluation of patient-administered tampon specimens for Chlamydia trachomatis and Neisseria gonorrhoeae.
SN Taborzi, CK Fairley, SJ Chen et al. Sex Transm Dis 2000;27:133–7


Impact of switching laboratory tests on reported trends in Chlamydia trachomatis infections.

Detection of Chlamydia trachomatis in pregnant women by the Papanicolaou technique, enzyme immunoassay and polymerase chain reaction.

Multicenter evaluation of the AMPLICOR and automated COBAS AMPLICOR CT/NG tests for detection of Chlamydia trachomatis.

Chlamydial development is adversely affected by minor changes in amino acid supply, blood plasma amino acid levels and glucose deprivation.

Differential regulation of CD4 lymphocyte recruitment between the upper and lower regions of the genital tract during Chlamydia trachomatis infection.

T-cell epitopes in variable segments of Chlamydia trachomatis major outer membrane protein elicit serovarspecific immune responses in infected humans.

Candidiasis

Vaginal colonization by Candida in asymptomatic women with and without a history of recurrent vulvovaginal candidiasis.

Effects of reproductive hormones on experimental vaginal candidiasis.

Evaluation of the Oricult-N dipslide for laboratory diagnosis of vaginal candidiasis.

Clonal and spontaneous origins of fluconazole resistance in Candida albicans.

Mechanisms of the proinflammatory response of endothelial cells to Candida albicans infection.

Bacterial vaginosis

Bacterial vaginosis.

Metronidazole to prevent preterm delivery in pregnant women with asymptomatic bacterial vaginosis.

Pre-term labor associated with bacterial vaginosis.
F Calderas, B Nievies, A Quintana. Anaerobe 1999;5:403–4
Trichomoniastis

Resistance of Trichomonas vaginalis to metronidazole: report of the first three cases from Finland and optimization of in vitro susceptibility testing under various oxygen concentrations.

Antigenicity of Trichomonas vaginalis heat-shock proteins in human infections.

Pelvic inflammatory disease

Pelvic inflammatory disease—an evidence-based approach to diagnosis.

Influence of human immunodeficiency virus infection on pelvic inflammatory disease.

Direct medical cost of pelvic inflammatory disease and its sequelae: decreasing but still substantial.

Syphilis and other treponematoses

Unraveling the Tuskegee Study for untreated syphilis.

Nodular tertiary syphilis mimicking granuloma annulare.

Social network method for endemic foci of syphilis: a pilot project.
R Rothenberg, L Kinbruch, R Lewishardy et al. Sex Transm Dis 2000;27:12–8

Geographic variation of HIV infection in childbearing women with syphilis in the United States.
EH Komans, M Sternberg, M Gwinn et al. AIDS 2000;14:279–88

HIV prevalence in patients with syphilis, United States.

From the CDC—syphilis elimination: history in the making—opening remarks.

Genital herpes and public health: addressing a global problem.
L Corey, HH Handsfield. JAMA 2000;283:791–4

Reactivation of genital herpes simplex virus type 2 infection in asymptomatic seropositive persons.

Herpes simplex virus type 2 shedding in human immunodeficiency virus-negative men who have sex with men: frequency, patterns and risk factors.

Editorial response: Asymptomatic herpes simplex virus shedding and Russian roulette.

Human immunodeficiency virus infection and genital ulcer disease in South Africa: the herpetic connection.

Medical care expenditures for genital herpes in the United States.

Herpes simplex virus DNA in amniotic fluid without neonatal infection.

Herpes simplex virus infection of the uterine cervix—relationship with a cervical factor?

The herpesvirus proteases as targets for antiviral chemotherapy.

Monoclonal antibodies suitable for type-specific identification of herpes simplex viruses by a rapid culture assay.

 Establishment of latent herpes simplex virus type 1 infection in resistant, sensitive and immunodeficient mouse strains.

Herpes simplex virus infection blocks events in the G1 phase of the cell cycle.
B Song, JZ Lie, KC Yeh, DM Knipe. Virology 2000;267:326–34
A rule for MHC class 1 down-regulation in NK cell lysis virus-infected cells.

Virus-induced neuronal apoptosis blocked by the herpes simplex virus latency-associated transcript.
GC Pering, C Jones, J Ciacciazzella et al. Science 2000;287:1500–2

Herpes simplex virus type-1 and -2 pathogenesis is restricted by the epidermal basement membrane.

Mitochondrial distribution and function in herpes simplex virus-infected cells.

Antegrade transport of herpes simplex virus type 1 in cultured, dissociated human and rat dorsal root ganglion neurons.

The latency-associated transcript gene enhances establishment of herpes simplex virus type 1 latency in rabbits.

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