LETTERS TO THE EDITOR

CMV polyradiculopathy in AIDS—suggestions for new strategies in treatment

Cytomegalovirus (CMV) polyradiculopathy presenting as a cauda equina syndrome is now a well described disorder in AIDS and is estimated to occur in 2% of AIDS patients with neurological problems. Although dihydroxypropxymethylguanine (DHPG, ganciclovir) has been shown to be effective, the optimum dose and duration of induction therapy have not been determined. Trisodium phosphonoformate guanine (foscarnet) has been shown to be efficacious in the treatment of some of the other complications of CMV, such as retinitis, but there have been no reports of its use in the polyradiculopathy syndrome.

A 41 year old bisexual man, HIV seropositive since 1984, progressed to AIDS in August 1988 when he developed Pneumocystis carinii pneumonia. In April 1990 he was admitted with a two week history of increasing weakness and paraesthesia in the legs, urinary frequency and two episodes of faecal incontinence. On examination the lower limbs were hypotonic and weak: MRC grade 4 proximally and grade 3 on the right, grade 1 on the left distally. The knee and ankle jerks were absent and the plantar responses equivocal. There was a mild sensory deficit with reduction to pinprick sensation over the S dermatome on the left. Anal tone was reduced and the bladder was palpable to the umbilicus. Examination of the upper limbs and cranial nerves was normal. There was no evidence of cytomegalovirus retinitis.

A thoracolumbar myelogram was normal. Cerebrospinal fluid examination showed 44 WBC, predominantly neutrophils, and the protein was 1.7 g/l. No organisms were cultured. Cytological examination and syphilis serological tests were negative. Nerve conduction tests on the lower limbs were consistent with a lumbar radiculopathy. A presumptive diagnosis of CMV polyradiculopathy was made and treatment was started with ganciclovir 10 mg/kg/day, 72 hours after admission. Zidovudine was stopped. Despite three weeks of treatment he continued to deteriorate and developed complete flaccid paralysis of his legs and required a suprapubic urinary catheter in situ. Empirically, foscarnet (57 mg/kg TID) was substituted for the ganciclovir. Zidovudine was reinstalled initially at a dose of 300 mg gradually increasing to 1000 mg daily.

The first signs of improvement were noted after two weeks of treatment with foscarnet. Over the next five weeks clinical improvement continued. However, owing to deteriorating renal function after 7 weeks of treatment with foscarnet, ganciclovir at a reduced maintenance dose of 3 mg/kg/day (5 out of 7 days) was reinstalled. This has continued to be self administered at home via a Hickman line. Zidovudine had been discontinued 4 weeks after it was restarted because of anaemia.

Five months after the initial presentation, the patient is mobile around his home using crutches but still requires an indwelling urinary catheter. The most prominent deficit is a left foot drop and absent ankle reflexes.

We report this case to highlight four issues regarding treatment of CMV polyradiculopathy in AIDS patients: firstly, substantial improvement is possible in this condition. Our patient improved from having a complete flaccid paralysis to being mobile with crutches. Secondly, unless the patient’s general condition dictates otherwise, a deterioration in spite of treatment should not be a deterrent to the continuation of anti CMV therapy. With regards to drug dosages and the necessity for maintenance treatment, much of the experience from CMV retinitis has been extrapolated to CMV polyradiculopathy. A review of the literature shows that induction therapies of ganciclovir 5–10 mg/kg/day for between 10 to 21 days are being used. This case suggests that high dose induction therapy of anti-CMV treatment should be continued at least until there is no further neurological improvement. Finally, although it is difficult to attribute improvement in our patient specifically to foscarnet, this may be an effective alternative to ganciclovir in CMV polyradiculopathy as in, for example, CMV retinitis. Furthermore, as foscarnet is less myelotoxic than ganciclovir, zidovudine therapy may be continued. Foscarnet should be considered as alternative therapy in cases where no improvement has occurred after two to three weeks of treatment with ganciclovir or in cases intolerant of ganciclovir. Clearly, further studies comparing the efficacy of these two therapies are required.

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We have previously reported a rise in the number of cases of sexually transmitted diseases (STDs) in Zambia from 1983 to 1987 and expressed concern over the exponential increase in cases of chancroid and syphilis in Zambia: 1987–89.1

As a follow-up, we reviewed and analysed monthly STD reports from clinics under the National STD Control Programme and now report STD trends from 1987 to 1989.

The table shows the number of STD clinics, annual STD cases and the four commonest STDs. In spite of the increase in the number of clinics from 28 to 40, there has been a decline in annual cases of STDs after 1987. Although the fact that nearly all clinics reported a decline suggests a genuine decline in STDs rather than just declining attendance at the clinics, it would still be useful to find out whether private practitioners and hospitals which do not report STDs to the National STD Control Programme have also seen a decline in STDs before crediting the decline entirely to the ongoing campaigns by the STD and AIDS Control Programmes.

Gonorrhoea, chancroid, syphilis and trichomoniasis are the four commonest STDs in decreasing order of frequency (table). Although gonorrhoea has previously been reported,1,2 and still continues, to be the commonest STD in Zambia, it is pertinent to note that following the exponential upsurge in cases of chancroid and syphilis in 1987,3 the incidence of genital ulceration has outstripped that of genital discharge/urethritis: genital ulcers accounted for 59% of 10,089 new cases of STD at the University Teaching Hospital, Lusaka, in 1989; and the annual cases of chancroid and syphilis combined, which previously were much less than gonorrhoea, have since surpassed those of gonorrhoea. As genital ulceration has been shown to be a risk factor for transmission3 and acquisition of HIV,4,5 the relative rise in genital ulceration is a cause for concern. Further work is required in order to elucidate factors underlying these trends.

In view of epidemiologic similarities between conventional STDs and HIV infection, the high incidence of STDs is an indicator of continued sexual transmission of HIV. And given the high prevalence of HIV among STD patients6,7 and the impact of concomitant HIV infection on conventional STDs,8 the current STD trends have serious implications for both the STD and AIDS control programmes.

**Table. Annual STD cases and the four commonest STDs in Zambia**

<table>
<thead>
<tr>
<th>Year</th>
<th>Total Cases</th>
<th>No. STD Clinics</th>
<th>Gonorrhoea</th>
<th>Chancroid</th>
<th>Syphilis</th>
<th>Trichomoniasis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1987</td>
<td>109,496</td>
<td>28</td>
<td>27,676</td>
<td>21,569</td>
<td>16,571</td>
<td>6,547</td>
</tr>
<tr>
<td>1988</td>
<td>101,286</td>
<td>38</td>
<td>25,705</td>
<td>17,486</td>
<td>19,972</td>
<td>5,039</td>
</tr>
<tr>
<td>1989</td>
<td>84,180</td>
<td>40</td>
<td>20,581</td>
<td>14,569</td>
<td>13,364</td>
<td>5,995</td>
</tr>
</tbody>
</table>

*Figures in brackets denote percentage of total annual cases.


Sexually transmitted infections in selected high risk populations in Cameroon

The renewed interest in sexually transmitted diseases (STDs) lies both in their increasing incidence and morbidity associated with them, as well as the fact that they enhance transmission of the human immunodeficiency virus (HIV).3,4

We wanted to study the prevalence of *Chlamydia trachomatis*, *Neisseria gonorrhoea*, syphilis and HIV infections in parturients, pregnant women and STD clinic attenders in Cameroon in order to develop a targeted STD/HIV intervention programme.

The study was carried out from March to May 1990, and August to October 1990. Cervical specimens were taken from 69 randomly selected parturients in the Laquitnique Hospital, Douala. We also recruited 192 patients (101 female and 91 male) from the STD clinic in Elig-Esson, Yaounde. Urethral samples were obtained and stored using the appropriate swabs and media (Abbott Laboratories, Wiesbaden, Germany). We tested 135 STD patients for *Chlamydia trachomatis* infection (69 men and 66 women), 120 (66 women and 54 men) for *Neisseria gonorrhoea* while only (47 women and 46 men) agreed to HIV testing.

In addition, 350 pregnant women attending the central antenatal clinic in Yaounde were recruited. The 192 STD patients and the 350 pregnant women were tested for antibodies to *Treponema pallidum*.

*Chlamydia* and gonorrhoea antigen detection were performed with commercial enzyme-immunoassays (*Chlamydialides* and Gonozyme from Abbott Laboratories, Wiesbaden). Anti-HIV antibody screening was performed with the Abbott recombinant HIV-1/2 enzyme immuno-assy. Positive samples were confirmed on line immuno assay and HIV-1 western blotting (Organon Teknika, Teknika, Turnhout, Belgium). Syphilis testing was done by the haemagglutination test (TPHA) obtained from Behring, Marburg, Germany (Cellognost Syphilis H). The results obtained are shown in the table. Sex prevalence among the STD patients for

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