Successful Resolution of Progressive Multifocal Leukoencephalopathy after Combination Therapy with Cidofovir and Cytosine Arabinoside

Str.—We read the interesting article by Blick et al. [1]. We challenge the conclusions made by the authors. Blick et al. point out that improvement in their patient’s condition was not related to antiretroviral therapy because progressive multifocal leukoencephalopathy (PML) developed 3 months after this treatment was applied. Certainly some opportunistic infections, such as cytomegalovirus retinitis, can develop soon after antiretroviral therapy is started [2]. However, this particular therapy can prevent the development or relapse of cytomegalovirus retinitis [3]. Therefore, the diagnosis of opportunistic infections in the early weeks of antiretroviral treatment does not necessarily mean this treatment will fail in the long term. The immune system needs more time to reconstitute itself [4], and the effect of antiretroviral therapy on opportunistic infections might be observed only after improvement of immune status.

Some investigators [4] observed improvement in the outcome of PML after antiretroviral therapy. We also observed extended survival in 2 patients who received this treatment (2 and 4 years of follow-up) and were previously diagnosed with PML by cerebral biopsy. The effect of cytosine arabinoside (ara-C) on PML has not yet been proved to be favorable [5]. However, Blick et al. suggest that ara-C and cidofovir, not antiretroviral therapy, caused improvement in their patient’s condition. We believe that this conclusion is unfounded. Perhaps they should have suggested that the combination of ara-C, cidofovir, and antiretroviral therapy had a favorable effect.

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References


Practice Guidelines for Evaluating New Fever in Critically Ill Adult Patients

Str.—In the excellent article [1] recently published in Clinical Infectious Diseases, O’Grady et al. comment on numerous non-infectious causes of fever, including 2 types of hyperthermia, malignant hyperthermia and neuroleptic malignant syndrome. However, they fail to point out that hyperthermia is a common finding in intensive care unit patients who do not have either of these 2 distinct conditions. Patients may be unable to control their temperature because of a variety of central and peripheral mechanisms [2]. In this era of increasing antimicrobial resistance, attention to this entity might allow less use of antibiotics for patients with “hyperthermia” and not “fever,” who simply need to have heat dissipation increased by simple measures.

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References


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Amoxicillin versus Sparfloxacin in the Treatment of Presumed Pneumococcal Pneumonia

Str.—I believe that the conclusions reached by Aubier et al. [1] in a recent trial comparing amoxicillin with sparfloxacin for the treatment of presumed pneumococcal pneumonia were flawed, as was their presentation of safety results.

Equivalent therapeutic outcomes were achieved with both regimens. This result, rather than favoring sparfloxacin, argues that the added spectrum of activity of sparfloxacin, although possibly important for certain nonpneumococcal pathogens in
selected patients, was on balance of no advantage for the study population. In view of the higher cost of sparloxacin and the risk of emergence of fluoroquinolone-resistant organisms with more widespread use of these compounds, the observed therapeutic equivalence should be taken as an argument for preferring amoxicillin over sparloxacin, rather than the reverse, so long as sparloxacin offers no clear safety advantage.

Yet in their study, Aubier et al. found that sparloxacin apparently did not offer a safety advantage, in spite of their misleading emphasis on the higher incidence of gastrointestinal side effects associated with amoxicillin. Close reading of the text reveals that discontinuation of the study drug for adverse effects was twice as common with sparloxacin as with amoxicillin, and that one-half of the discontinuations involving sparloxacin (but none involving amoxicillin) were for gastrointestinal symptoms.

Therefore, this study can be legitimately interpreted as providing reassurance that even in locales where historically the prevalence of penicillin-resistant pneumococci is high, high doses of amoxicillin remain an effective and well-tolerated regimen for mild to moderately severe presumed pneumococcal pneumonia in otherwise healthy adults and that newer, extended-spectrum agents are not necessary in this context.

It should be noted that the study was funded by Rhône DPC (Antony, France), the manufacturer of sparloxacin, who would stand to benefit from the authors’ quite different conclusions. The infectious diseases community and the general public are not well served by recommendations to use newer, more expensive agents on the basis merely of demonstrated equivalence with traditional regimens. This disservice is all the greater when study results are presented in an unbalanced fashion that favors the sponsor’s product. It is disappointing that the author of the editorial response to this study did not comment on these issues [2].

References


Reply

SIR—The points raised by Johnson are well taken and supplement rather than conflict with my comments [1]. I hope that he did not interpret my views as advocating the use of the new fluoroquinolones for treatment of community-acquired pneumonia in preference to more traditional therapies.

The issue of pharmaceutical industry sponsorship of clinical trials involves most medical specialties. It is simply a fact of life that studies of new therapeutic agents have some component of industrial sponsorship or involvement, and *Clinical Infectious Diseases* and other specialty journals reflect this.

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*Burkholderia pseudomallei* Infections

SIR—I am always pleased to see anything that may heighten physicians’ awareness of melioidosis and *Burkholderia pseudomallei*, such as the 2 articles recently published in *Clinical Infectious Diseases* [1, 2]. However, I would like to add a few comments to these reports.

Dorman et al. [1] incorrectly claim that our study from northeast Thailand [3] supports the use of cefazidime plus trimethoprim-sulfamethoxazole to treat severe melioidosis. In fact, cefazidime monotherapy was used in this study partly because of concern about bactericidal antagonism between cefazidime and trimethoprim-sulfamethoxazole [4]. Cefixime has not been evaluated for use in the treatment of melioidosis, despite evidence of in vitro activity, and for children aged 11 years, I would advocate the use of amoxicillin/clavulanate treatment for 20 weeks [5]. In their table 1, Dorman et al. [1] usefully summarize confirmed cases of melioidosis apparently indigenous to the Americas and the Caribbean. I can also provide independent confirmation of the identity of the isolate from Guadeloupe that was reported by Pérez et al. [6]. Using biochemical testing and the antibiogram at the Central Public Health Laboratory (Colindale, UK), we found it to be indistinguishable from clinical isolates of *B. pseudomallei* from Southeast Asia and northern Australia. Clearly, further studies...