Table 1. (*Continued*)

Immunosuppressive agents/conditions	APACHE II score	Antecedent antibiotic[‡]	Concomitant infection (species, no. of cultures positive/total)	Treatment	Duration of treatment (d)	Outcome
CyspA + Prd	15	Ctax	Liver abscess (*E. gallinarum*)	Amp/Sulb	14	Survived
CyspA + Prd	16	Cpfx	None	Amp/Sulb	12	Survived
Tacrolimus + Prd	NA[§]	Czid, Vm	Primary peritonitis (*E. casseliflavus*)	Amp + Gm	15	Survived
Cirrhosis	15	. . .	Bacteremia (*Acinetobacter lwoffii,* 2/2)	Vm, then Amp/Sulb	14	Survived
CyspA + Prd	12	. . .	Bacteremia (*Aeromonas hydrophila,* 2/2)	Vm, then Pip/Taz	14	Survived
Neutropenia following chemotherapy	NA[§]	Czid, Vm	Bacteremia (*Escherichia coli, Klebsiella pneumoniae, Enterobacter cloacae,* 1/2)	Czid + Gm + Vm	6	Died
CyspA + Prd	7	Czid, Vm, Cpfx, TMP-SMZ	None	Vm + Cpfx	14	Died
Prd	9	Imi	Bacteremia (*Corynebacterium* species, 6/10)	Cpfx + Cm	10	Survived

References

1. Pompei R, Lampis G, Berlutti F, Thaller MC. Characterization of yellow-pigmented enterococci from severe human infections. J Clin Microbiol **1991**;29:2884–6.
2. Patterson JE, Sweeney AH, Simms M, et al. An analysis of 110 serious enterococcal infections. Epidemiology, antibiotic susceptibility, and outcome. Medicine (Baltimore) **1995**;74:191–200.
3. Toye B, Shymanski J, Bobrowska M, Woods W, Romotar K. Clinical and epidemiologic significance of enterococci intrinsically resistant to vancomycin (possessing the van C genotype). J Clin Microbiol **1997**;35:3166–70.
4. Iwen PC, Kelly DM, Linder J, Hinrichs SH. Revised approach for identification and detection of ampicillin and vancomycin-resistance in Enterococcus species by using MicroScan panels. J Clin Microbiol **1996**;34:1779–83.
5. Cartwright CP, Stock F, Fahle GA, Gill VJ. Comparison of pigment production and motility tests with PCR for reliable identification of intrinsically vancomycin-resistant enterococci. J Clin Microbiol **1995**;33:1931–3.

Worsening of Endogenous *Candida albicans* Endophthalmitis During Therapy with Intravenous Lipid Complex Amphotericin B

Endogenous fungal endophthalmitis is a potentially blinding disease that occurs in the setting of immunosuppression, gastrointestinal surgery, and long-term antibiotic therapy [1]. In most cases, the treatment of choice is intravenous amphotericin B, but therapy is often limited by nephrotoxicity [1, 2]. The lipid complex and liposomal preparations of amphotericin B have been developed to decrease this complication, but their efficacy in treating fungal endophthalmitis is not known. We describe a patient with endogenous *Candida albicans* endophthalmitis that progressed after intravenous antifungal therapy was changed from fluconazole to lipid complex amphotericin B.

A 39-year-old woman with inflammatory bowel disease was transferred to our hospital on 11 June 1997 for management of polymicrobial sepsis due to *Klebsiella pneumoniae* and *Enterococcus faecalis.* She was treated with intravenous vancomycin, gentamicin, and imipenem. When subsequent blood cultures yielded coagulase-negative *Staphylococcus* and *C. albicans,* fluconazole therapy was added.

Correspondence: Dr. Myron S. Cohen, 547 Burnett-Womack, CB #7030, Chapel Hill, North Carolina 27599-7030.

Clinical Infectious Diseases 1999;28:1177–8

1058–4838/99/2805–0047$03.00

The patient was initially examined by the ophthalmology service on 17 June. Her vision was 20/20 in both eyes. A dilated fundus examination was remarkable for findings of cotton-wool spots and white-centered retinal hemorrhages bilaterally. The left eye had vitreous opacities consistent with fungal endophthalmitis, and fluconazole therapy was continued. Reexamination on 23 June revealed resolving white-centered hemorrhages and stable-appearing fungal infiltrates.

On 24 June, a peripheral blood culture yielded *Candida* (*Torulopsis*) *glabrata,* and her intravenous antifungal medication was changed from fluconazole to lipid complex amphotericin B (Abelcet; Liposome, Princeton, NJ) to treat against both fungal organisms. The lipid complex formulation was used because of a creatinine level elevation from 0.7 to 1.5 mg/dL during the previous 2 weeks of antimicrobial therapy. Antibacterial therapy was discontinued.

The patient noticed decreasing vision in the left eye. She was reexamined on 3 July, and visual acuity was 20/25 in the right eye and 20/200 in the left eye. Slit-lamp examination of the left eye was remarkable for the finding of severe anterior segment inflammation, including cells in the anterior vitreous. A dilated fundus examination of the left eye showed moderate vitreous haze and a fluffy exudate along the inferotemporal arcade near the disk. A vitrectomy was performed, and the patient received intravitreal ceftazidime (2 mg), vancomycin (1 mg), and amphotericin B (5 μg). No fungi were seen in the undiluted KOH stain, but the vitreous fluid cultures yielded *C. albicans.*

The patient's condition improved clinically, and she was discharged 1 week later while being treated with oral fluconazole, topical prednisolone acetate (1%), and topical scopolamine (0.25%). She was subsequently lost to follow-up.

This case raises many important questions. The first question concerns the appropriate interval of follow-up for patients being treated systemically for *C. albicans* endophthalmitis. Although most sources indicate that follow-up should be based on clinical impression, a recent multicenter trial had follow-up intervals of 1 week without progression of candidal retinochoroiditis [3].

Another question concerns the agent of choice in fungal endophthalmitis. Intravenous amphotericin B, alone or with flucytosine, has been shown to be effective in treating *C. albicans* endophthalmitis and is currently the treatment of choice [1]. Intravitreal injections and vitrectomy may also be required for cases with mild to severe vitreous involvement [1]. Recently, oral fluconazole has been investigated for use in the treatment of fungal endophthalmitis, but it is useful only against *C. albicans* because *Candida krusei, C. glabrata, Fusarium* species, and *Aspergillus* species are resistant [2]. In a direct comparison, Filler et al. [4] found intravenous amphotericin B to be superior to intravenous fluconazole in the treatment of disseminated candidiasis and endophthalmitis in a rabbit model.

The recently developed lipid complex and liposomal preparations of amphotericin B have not yet been thoroughly evaluated for treatment of ocular infections. In a rabbit model of *C. albicans* endophthalmitis, Liu et al. [5] showed that intravitreal liposomal amphotericin B was well tolerated, but there was decreased efficacy compared with that of regular amphotericin B.

This case suggests that unlike the regular formulation, the lipid complex preparation of amphotericin B may not always be sufficient to treat *C. albicans* endophthalmitis. This circumstance may be due to both poor intraocular penetration and decreased intraocular efficacy.

Steven R. Virata, Jan A. Kylstra, Justin C. Brown, David A. Wohl, and Myron S. Cohen

Departments of Ophthalmology and Medicine, University of North Carolina School of Medicine, Chapel Hill, North Carolina

References

1. Samiy N, D'Amico DJ. Endogenous fungal endophthalmitis. Int Ophthalmol Clin **1996**;36(3):147–62.
2. Luttrull JK, Wan WL, Kubak BM, et al. Treatment of ocular fungal infections with oral fluconazole. Am J Ophthalmol **1995**;119:477–81.
3. Donahue SP, Greven CM, Zuravleff JJ, et al. Intraocular candidiasis in patients with candidemia: clinical implications derived from a prospective multicenter study. Ophthalmology **1994**;101:1302–9.
4. Filler SG, Crislip MA, Mayer CL, Edwards JE. Comparison of fluconazole and amphotericin B for treatment of disseminated candidiasis and endophthalmitis in rabbits. Antimicrob Agents Chemother **1991**;35:288–92.
5. Liu KR, Peyman GA, Khoobehi B. Efficacy of liposome-bound amphotericin B for the treatment of experimental fungal endophthalmitis in rabbits. Invest Ophthalmol Vis Sci **1989**;30:1527–34.

A DNA-Based Probe for Differentiation of *Giardia lamblia* Group A and B Isolates from Northern India

Giardia lamblia (also known as *Giardia intestinalis*) is considered an important cause of diarrhea and malabsorption worldwide [1]. *G. lamblia* strains responsible for acute and chronic diarrhea cause malabsorption of nutrients and fats in some adults and children. In contrast, other adults and children infected with *G. lamblia* strains are asymptomatic carriers. At present, the factors responsible for the development of diarrhea are poorly understood, and it is still not clear whether the characteristics of the parasite, the host, or both are responsible for the varied clinical manifestations [2].

A number of techniques have been used thus far to determine the heterogeneity among *G. lamblia* isolates. Isoenzyme and molecular biological studies have separated *G. lamblia* strains into two groups, group A (also called Polish) and group B (also called Belgian) [3, 4]. The two *G. lamblia* groups appear no more related to each other (homologous genes show 81%–89% identity) than are the protozoan parasites *Entamoeba histolytica* and *Entamoeba*

Reprints or correspondence: Dr. N. K. Ganguly, Indian Council of Medical Research, New Delhi, 110029, India (icmrhqds@sansad.nic.in).

Clinical Infectious Diseases 1999;28:1178–80

1058–4838/99/2805–0048$03.00