In addition to a significant decrease over time in the overall prevalence of *P. carinii* pneumonia at autopsy, these observations document unequivocally the emergence of atypical, tissue-destructive *P. carinii* pneumonia in patients with advanced AIDS. Its association with extrapulmonary pneumocystosis suggests that tissue necrosis may be required for dissemination of this otherwise strictly intraalveolar opportunistic pathogen [5].

**Table 1.** Presentation of AIDS-associated *Pneumocystis carinii* pneumonia at autopsy at the University Hospital of Frankfurt/Main, Germany, 1982–1992.

<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td><strong>No. (%) of cases</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute*</td>
<td>1 (25)</td>
<td>9 (23)</td>
<td>5 (14)</td>
</tr>
<tr>
<td>Chronic†</td>
<td>3 (75)</td>
<td>30 (77)</td>
<td>16 (46)</td>
</tr>
<tr>
<td>Atypical‡</td>
<td>...</td>
<td>...</td>
<td>14 (40)§</td>
</tr>
<tr>
<td><strong>Total no. of cases/no. of autopsies (%)</strong></td>
<td>4/13 (31)</td>
<td>39/112 (35)</td>
<td>35/234 (15)§</td>
</tr>
</tbody>
</table>

* Characterized by signs of acute alveolar damage, sparse interstitial infiltrates of predominantly mononuclear origin, and typical foamy intraalveolar exudate containing numerous cyst forms of the organism.
† Characterized by signs of chronic alveolar damage with interstitial and/or intraalveolar proliferation of fibroblasts, interstitial mononuclear infiltrates, and foamy exudate with cyst forms.
‡ Characterized by mostly areactive tissue necrosis, cavitation, or honeycombing, variably surrounded by fibrous tissue, mononuclear cells, focal calcifications, or small, granuloma-like lesions with variable extent of fibrosis and/or calcification. In both instances, *P. carinii* infection is found within the lesions and in the neighboring parenchyma.
§ *P* < .001 (by the Kruskal-Wallis H test).

for a mean of 16 months before death (range, 5–28 months). Of importance is the fact that atypical *P. carinii* infection was diagnosed during life in only one-half of the cases.

## Failure of Amphotericin B Lipid Complex in the Treatment of Cutaneous Leishmaniasis

Cutaneous leishmaniasis in Central and South America is caused most commonly by *Leishmania braziliensis*, *Leishmania guyanensis*, *Leishmania panamensis*, and *Leishmania mexicana*, and the recommended treatment is one of the pentavalent antimonial compounds (meglumine antimoniate or sodium stibogluconate) [1]. Recent reports of visceral leishmaniasis (kala-azar) have suggested that treatment with lipid formulations of amphotericin B may be as or more effective than antimony [2, 3]; one study demonstrated that visceral leishmaniasis can be cured with as few as 5 days of treatment with amphotericin B lipid complex (Abelcet; The Liposome Co., Princeton, NJ) [4]. In contrast, the use of lipid amphotericin B formulations for cutaneous disease has been reported rarely. One report describes the efficacy of liposomal amphotericin B (AmBisome; Vestar, San Dimas, CA) at a dosage of 1.5 mg/(kg·d) for 2 weeks for the treatment of an antimony-resistant case of cutaneous leishmaniasis acquired in the Mediterranean (the species of *Leishmania* was not reported). However, the patient also received 2.5 g of amphotericin B after completion of therapy with AmBisome [5]. We describe a patient with cutaneous leishmaniasis in whom treatment with Abelcet failed and who was cured subsequently with sodium stibogluconate (Pentostam; Wellcome Foundation, London, UK).

A 25-year-old male American soldier stationed in Panama presented originally with a 2-month history of a nonhealing ulcer over his left first metacarpophalangeal joint. Cultures of biopsy specimens of the ulcer and of an excised enlarged lymph node located on his left forearm yielded *L. braziliensis*. The patient was treated with Abelcet, 3 mg/(kg·d), for 16 days (total dose, 4.3 g). By the end of treatment, the patient’s ulcer had regressed dramatically, with almost complete reepithelialization, and he returned to duty in Panama. Approximately 3 weeks after treatment with Abelcet had been discontinued, the patient noted breakdown of his previously healed lesion. He was referred to Walter Reed Army Medical Center (Washington, D.C.), where examination of the left first metacarpophalangeal joint revealed a 1-cm area of hyperpigmentation with central ulceration. Mild adenopathy was noted in the left axilla.

**References**

Biopsy of the ulcerated site revealed superficial and deep chronic perivascular inflammatory infiltrate without visible amastigotes, but culture of the biopsy specimen yielded *L. braziliensis*. Sodium stibogluconate, 20 mg/(kg·d) for 10 days vs. 20 days, was administered as part of an ongoing protocol. During treatment, small satellite lesions developed around the ulcer, and two vesicular lesions arose at the base of the ulcer. By the end of treatment, the site of the lesion had improved markedly, and a follow-up examination 2 months after completing treatment demonstrated complete reepithelialization and no clinical evidence of recurrence.

Concerns regarding toxicity and reports of increasing resistance to the pentavalent antimonial compounds have led to a search for alternative treatment regimens for leishmaniasis. Lipid formulations of amphotericin B accumulate in the liver and spleen [6], which are the primary sites of infection for visceral leishmaniasis. With cutaneous disease, however, the site of infection is the dermis [7]. Panosian et al. [8] reported that lipid-intercalated amphotericin B has no effect on cutaneous leishmaniasis in immunocompetent mice and only a slight effect in the immunodeficient BALB/c strain. More recently, Yardley and Croft [9] demonstrated in a murine model that AmBisome reduced the size of lesions in experimental cutaneous leishmaniasis in a dose-dependent manner; however, all mice relapsed, suggesting that AmBisome had a suppressive rather than curative effect.

Our experience with the use of Abelcet for the treatment of cutaneous leishmaniasis in this single case concurs with the lack of curative effect seen in the murine model when lipid formulations of amphotericin B have been used to treat cutaneous leishmaniasis. On the basis of the available published data, we suggest that lipid formulations of amphotericin B are as yet insufficiently studied for the treatment of cutaneous disease and that, pending the accumulation of further data, the recommended therapy for New World cutaneous leishmaniasis continues to be one of the pentavalent antimonials. Whether higher doses, longer courses, or different lipid formulations of amphotericin B would prove to be more efficacious can only be surmised.

**Successful Treatment of Severe Cytomegalovirus Infection with Ganciclovir in an Immunocompetent Host**

Severe life-threatening infection due to cytomegalovirus (CMV) is rare in immunocompetent hosts. A recent review of the medical literature revealed severe CMV disease in only 34 previously healthy individuals [1]. The use of specific antiviral therapy in these patients did seem to confer a survival advantage; however, there are no guidelines or recommendations for the use of such therapy in this context. We describe a previously healthy patient with severe CMV myocarditis, pneumonitis, and hepatitis whose condition responded favorably to a 2-week course of iv ganciclovir.

A 31-year-old male presented with fever, abdominal pain, jaundice, cough, palpitations, and shortness of breath. He was tachypneic (respiratory rate, 32) with fever (temperature, 38.7°C), hypotension (blood pressure, 90/70 mm Hg), tachycardia (pulse, ≥200/min) with atrial fibrillation, elevated jugular venous pressure, cardiomegaly, a third heart sound, bilateral basal crackles, and tender hepatomegaly. He had no significant medical history or family medical history, and he had never received blood products. He was married and had one child, and he was a nonsmoker.

A chest radiograph showed bilateral interstitial pulmonary infiltrates and a normal heart size and configuration. Retropertioneal lymphadenopathy was observed on abdominal CT scanning, but there were no detectable lymph nodes elsewhere. Results of laboratory studies revealed the following abnormalities: neutrophil count, 21.2 × 10⁹/L; lymphocyte count, 1.2 × 10⁹/L; and platelet count, 544 × 10⁹/L. A blood film showed toxic granulation. The prothrombin time was 19 seconds; plasma fibrinogen level, 7.73 g/L; serum sodium level, 128 mmol/L; albumin level, 28 g/L; aspartate aminotransferase level, 173 U/L; alanine aminotransferase level, 273 U/L; γ-glutamyl transferase level, 787 U/L; alkaline phosphatase level, 815 U/L; and total bilirubin level, 63 μmol/L. The

References