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Myositis Associated with a Newly Described Microsporidian, *Trachipleistophora hominis*, in a Patient with AIDS

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Microsporidia are zoonotic protozoa which were rare human pathogens prior to 1985, when *Enterocytozoon bieneusi* was described in human immunodeficiency virus-infected patients with chronic diarrhea. Another species, *Encephalitozoon* (*Septata*) *intestinalis*, is associated with diarrhea and chronic sinusitis, and approximately 25 cases have been reported in the literature. However, other microsporidial infections in human immunodeficiency virus-infected patients remain extremely rare. We report the first case of a *Pleistophora* sp.-like microsporidian infection presenting as a progressive severe myosotis associated with fever and weight loss. The organism was demonstrated by light microscopy and electron microscopy in corneal scrapings, skeletal muscle, and nasal discharge. Electron microscopy showed an electron-dense surface coat with "sunflare"-like projections surrounding all stages of development of meronts (two to four nuclei, dividing by binary fission), sporonts, and sporoblasts. Division of sporonts, in which sporonts separate from the thick outer coat, creating a sporophorous vesicle, is by binary fission, differentiating this organism from *Pleistophora* sp. The spore measures 4.0 by 2.5 μm and has a rugose exospore. A new genus and species, *Trachipleistophora hominis*, has been established for this parasite. The patient was treated with albendazole, sulfadiazine, and pyrimethamine, and the clinical symptoms resolved.

Microsporidia are obligate intracellular parasites which were first reported in association with human immunodeficiency virus (HIV) infection in 1985 (26). Since that time, there have been several descriptions of microsporidiosis in AIDS patients. The majority of reports concern *Enterocytozoon bieneusi*, which invades primarily the small bowel enterocytes but has also been described in nasal and gall bladder epithelial cells. It has been associated with chronic diarrhea, malabsorption (13, 14, 27), acalculous cholecystitis, cholangitis (28), and rhinitis (12). *Encephalitozoon* (*Septata*) *intestinalis*, a more recently described microsporidian (2, 3, 16), causes disseminated infection and is the second most frequently described microsporidian in HIV-infected patients (14). We recently reported on a series of nine patients with *E. intestinalis* infections in whom diarrhea, chronic rhinosinusitis, and respiratory and urinary tract involvement were prominent features (10). Infection with *E. intestinalis* responds both clinically and microbiologically to treatment with the antiparasitic agent albendazole (10, 34), in contrast to *E. bieneusi*, against which the clinical and parasitological benefit of albendazole and metronidazole in uncontrolled trials has been variable (9, 11, 17). Albendazole also is effective in vitro against *Encephalitozoon cuniculi* (7).

Other microsporidial species have been described in HIV-infected patients. *E. cuniculi* has been associated with hepatitis (30), peritonitis (35), and nephritis (18, 19). *Encephalitozoon hellem* can cause disseminated infection (29, 33), keratoconjunctivitis (8, 33), interstitial nephritis (33), and chronic sinusitis (23, 29). There have been two reports of infections of human skeletal muscle caused by *Pleistophora*, a genus common in insects and cold-blooded vertebrates, especially fish (4, 5). The first case was reported in 1985 in a 20-year-old African American with fever, muscle wasting, and weakness who was thought to have AIDS (24). However subsequent HIV antibody tests were negative. More recently, Chupp et al. described a 33-year-old Haitian male with AIDS and a 2-month history of fever, myalgia, weakness, and a productive cough (6). Muscle biopsy suggested the presence of *Pleistophora* sp., which was confirmed by electron microscopy. Therapy with pyrimethamine and clindamycin was unsuccessful, and the myositis progressed to death within 3 weeks.

We report here the first case of a *Pleistophora*-like microsporidian infection involving skeletal muscle and, in addition, the nasal sinus cavities and conjunctiva. Cultures in several cell lines were established from spores purified from the muscle biopsy, and infection was established in athymic mice by intramuscular and intraperitoneal inoculations (22). Description of the parasite from culture and in mouse skeletal muscle and assignment of the parasite to a new genus and species are the subjects of a separate report (21). Here, we report the clinical presentation, light and electron microscopy findings, and response to therapy.

(The material in this report was presented, in part, orally at the 7th Annual Conference of the Australasian Society for HIV Medicine, Coolum, Australia, November 1995, by D.J.M. and, in part, orally at the Joint Meeting of the American Society of Parasitologists and the Society of Protozoologists, Tucson, Ariz., June 1996, by A.S.F.)

CASE REPORT

A 34-year-old commercial pharmacist known to be HIV infected was admitted to the hospital with incapacitating myalgias and proximal limb weakness. Concurrent problems were diplopia, lethargy, progressive weight loss, odynophagia, and a productive cough.

HIV infection was first noted some 9 years prior to admission, and homosexual contact was the mode of transmission.

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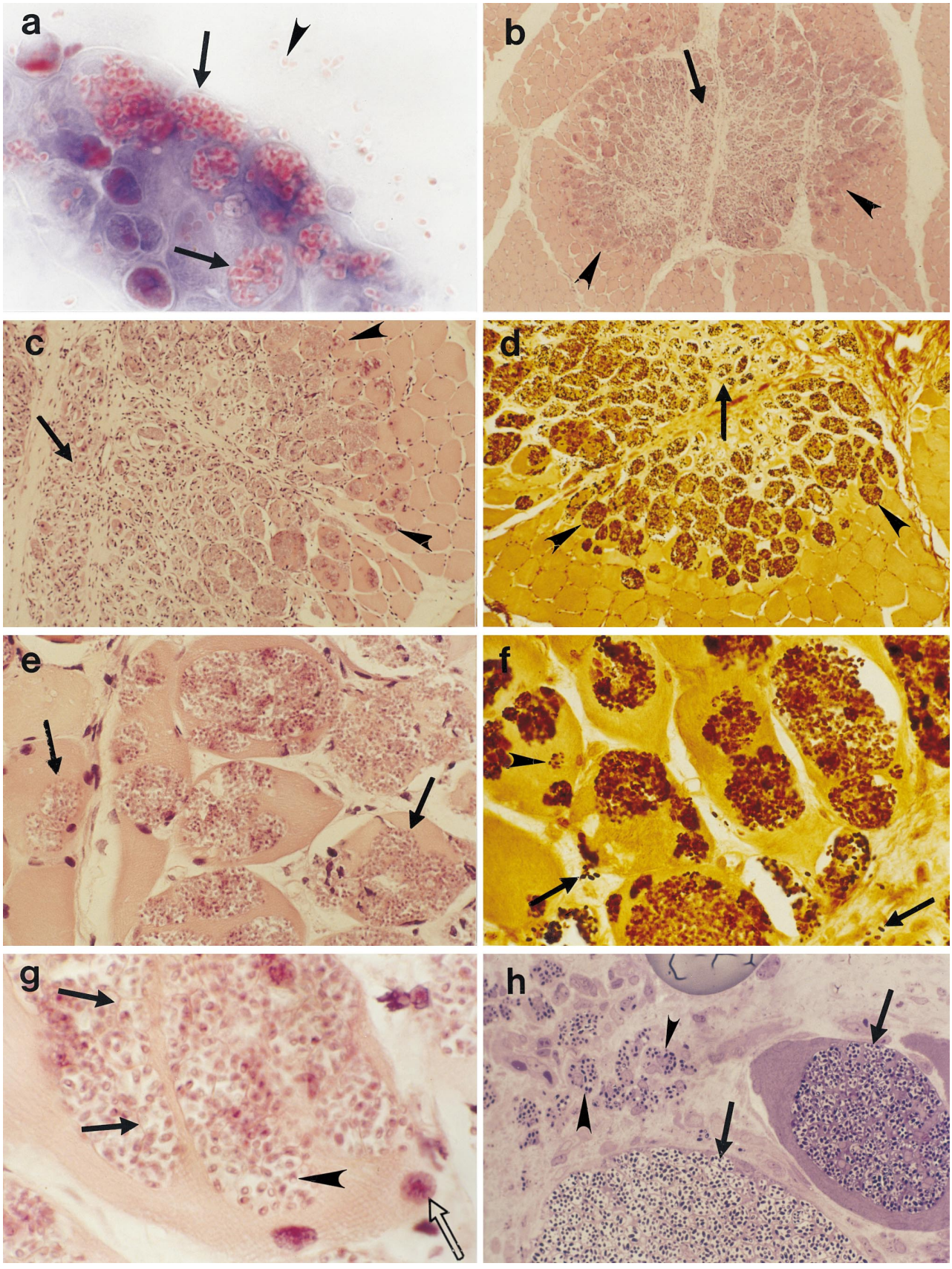

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Cryptococcal meningitis, his first AIDS-defining illness, was diagnosed 11 months before admission and treated successfully with fluconazole with complete resolution of the headache after 1 month of therapy. Fluconazole maintenance therapy was continued. Other HIV-related problems included recurrent oral candidiasis and seizures secondary to cryptococcal meningitis. He was known to be hepatitis B surface antigen positive, and there was a history of asthma in childhood.

The patient was well until myalgias began 3 months prior to admission with pain and stiffness of the thigh and forearm muscles. There was no headache or other symptoms to suggest recurrence of cryptococcal meningitis. Examination at that time revealed only mild proximal weakness. Creatinine kinase (CK) levels were persistently elevated, with values between 351 and 390 U/liter (normal, <130 U/liter). Diclofenac and amitryptiline were partially effective in controlling the pain. One month before admission, the patient developed bilateral keratoconjunctivitis, which improved with topical prednisolone and chloramphenicol. Corneal scrapings performed at another institution suggested the presence of a microorganism resembling a large microsporidial spore, but it could not be fully identified at the time.

The patient was admitted to the hospital in January 1995 and was extremely unwell, with progressive, persistent, severe muscle pain with intense, lancinating exacerbations and fevers to 39°C. He was immobilized because of pain and wasting and had lost 6 kg despite maintaining good oral food intake. Medications included fluconazole, cotrimoxazole, morphine, diclofenac, acyclovir, coloxyl with senna and chloramphenicol and prednisolone eye drops. He gave no history of recent overseas travel but had been to Europe, the United Kingdom, the United States of America, and Southeast Asia in the preceding decade. His most recent trip had been to Hawaii 2 years earlier. He kept a dog but no other animal as a pet. He did not consume raw or partly cooked meat or fish. He worked as a commercial pharmacist in a country town but had no exposure to farm animals or the native flora or fauna. He had a productive cough, but CXR and sputum cultures, including induced sputum, for *Pneumocystis carinii* and *Mycobacterium avium* complex organisms were negative.

On examination, his weight was 67 kg. There was marked muscle wasting with contractures of the elbows, wrists, and ankles. The major muscle groups were exquisitely tender. Power was reduced commensurate with the degree of wasting and discomfort. He complained of variable diplopia with horizontal separation of images and discomfort with eye movement, but no definite ocular nerve palsy or proptosis was detectable. There was also difficulty with swallowing. There was no evidence of abnormal reflexes, fasciculations, involuntary movements, neuropathy, skin rash, or arthropathy. The spleen was just palpable, and there was no lymphadenopathy or hepatomegaly. The body temperature was 39°C, with a sinus tachycardia of 124/min and a cardiac gallop rhythm. Respiration was normal.

Investigations revealed moderate pancytopenia, with hemoglobin level of 91 g/liter, a leukocyte count of 2.3×10^9/liter (neutrophils, 1.9; lymphocytes, 0.2), and a platelet count of 217×10^9/liter. The CK level was markedly elevated at 1,410 U/liter, and lactate dehydrogenase was 1,930 U/liter. Serum alkaline phosphatase, gamma glutamyl transpeptidase, and alanine transaminase levels were also elevated at 167, 193, and 106 U/liter, respectively. Serum bilirubin, electrolytes, and renal function were normal. Electrocardiographic findings were normal. Electromyography showed increased numbers of polyphasic units consistent with polymyositis. Results of investigations for autoimmune disease, including antinuclear antibody, serum complement, and smooth muscle, antimitochondrial, and gastric parietal cell antibodies, were negative or normal. Serum cryptococcal antigen was positive (titer, 1 in 1,024), consistent with the earlier diagnosis of cryptococcal meningitis. Serological tests for syphilis, toxoplasmosis, histoplasmosis, and coccidioidomycosis were negative.

A biopsy of the deltoid muscle was performed, and examination revealed the presence of structures suggesting the possibility of leishmaniasis or sarcocystis. Microsporidiosis was considered less likely, as the spores were approximately 4 μm long, significantly larger than other disseminated microsporidia described in humans. The muscle biopsy was reviewed in conjunction with W. Hartley (FRC Path, consultant pathologist, Taronga Park Zoo, Sydney, Australia), who suggested similarities to a zoonotic microsporidial infection. Concurrently, the electron microscopy was completed and confirmed the diagnosis of microsporidiosis. A second deltoid muscle biopsy, nasopharyngeal aspirate, corneal scraping for microscopy, and culture of fecal and urine specimens were obtained.

Treatment was commenced with a combination of albendazole (400 mg twice daily), sulfadiazine (1 g four times daily), pyrimethamine (50 mg daily), and folinic acid (7.5 mg daily). Within 4 days, the patient noted some improvement, with decreased pain, improved mobility, and defervescence, although he remained extremely weak. His body weight remained stable, and the CK fell to 491 U/liter. His pancytopenia remained stable. No fluctuations in liver function test or renal test results were associated with the progress of his infection or treatment. Recovery was impeded by nausea, emesis, and the development of probable staphylococcal pneumonia and cytomegalovirus retinitis. Sixty-five days following admission, the patient was discharged from the hospital. He was pain free and able to ambulate with assistance and had a stable weight of 53 kg and a normal CK level. His cough and diplopia had resolved. His odynophagia resolved, but his failure to gain weight necessitated percutaneous gastric feeding.

FIG. 1. (a) Conjunctival smear showing sporophorous vesicles containing spores and spore precursors in epithelial cells (arrows). Note the dispersed spores in the background showing the posterior vacuole (arrowhead) (modified trichrome stain; magnification: ×1,000, oil). (b) Lesion in skeletal muscle with central atrophied fibers (arrow) and outer fibers filled with basophilic spores and spore precursors (arrowheads) (H and E stain; magnification, ×20). (c) Lesion in skeletal muscle showing central atrophic fibers with a minimal inflammatory response (arrow) and an outer zone of muscle fibers containing basophilic aggregates of vacuoles (arrowheads) (H and E stain; magnification, ×100). (d) Lesion in skeletal muscle showing recognizable black spores (arrow) in central atrophic fibers and spores and brown spore precursors in peripheral skeletal muscle fibers (arrowheads) (Warthin-Starry stain; magnification, ×100). (e) Spores and spore precursors clearly visible in rounded spaces (arrows) within skeletal muscle fibers. Note that the fibers retain their shape and size while the sarcoplasm is replaced by vacuoles and that there is an absence of inflammatory cells (H and E stain; magnification, ×400). (f) Spores and larger brown spore precursors forming masses within skeletal muscle fibers. Free spores are visible in the adjacent connective tissues (arrow), and discrete early aggregates of spore precursors are visible in fibers (arrowhead) (Warthin-Starry stain; magnification, ×400). (g) Discrete walls of abutting sporophorous vesicles (arrows) in the sarcoplasm of skeletal muscle cells with their peripheral nuclei. The posterior vacuole and pink-stained anterior anchoring plate and sporoplasm are visible in individual spores (arrowhead). Note the peripheral nuclei of the skeletal muscle fiber (open arrow) (H and E stain; magnification, ×1,000; oil). (h) Sporophorous vacuoles containing spores and spore precursors in the virtually replaced sarcoplasm of skeletal muscle fibers (arrows). Spores are also visible in adjacent smaller cells, which electron microscopy showed to be macrophages (arrowheads) (toluidine blue-stained, resin-embedded section; magnification, ×400).

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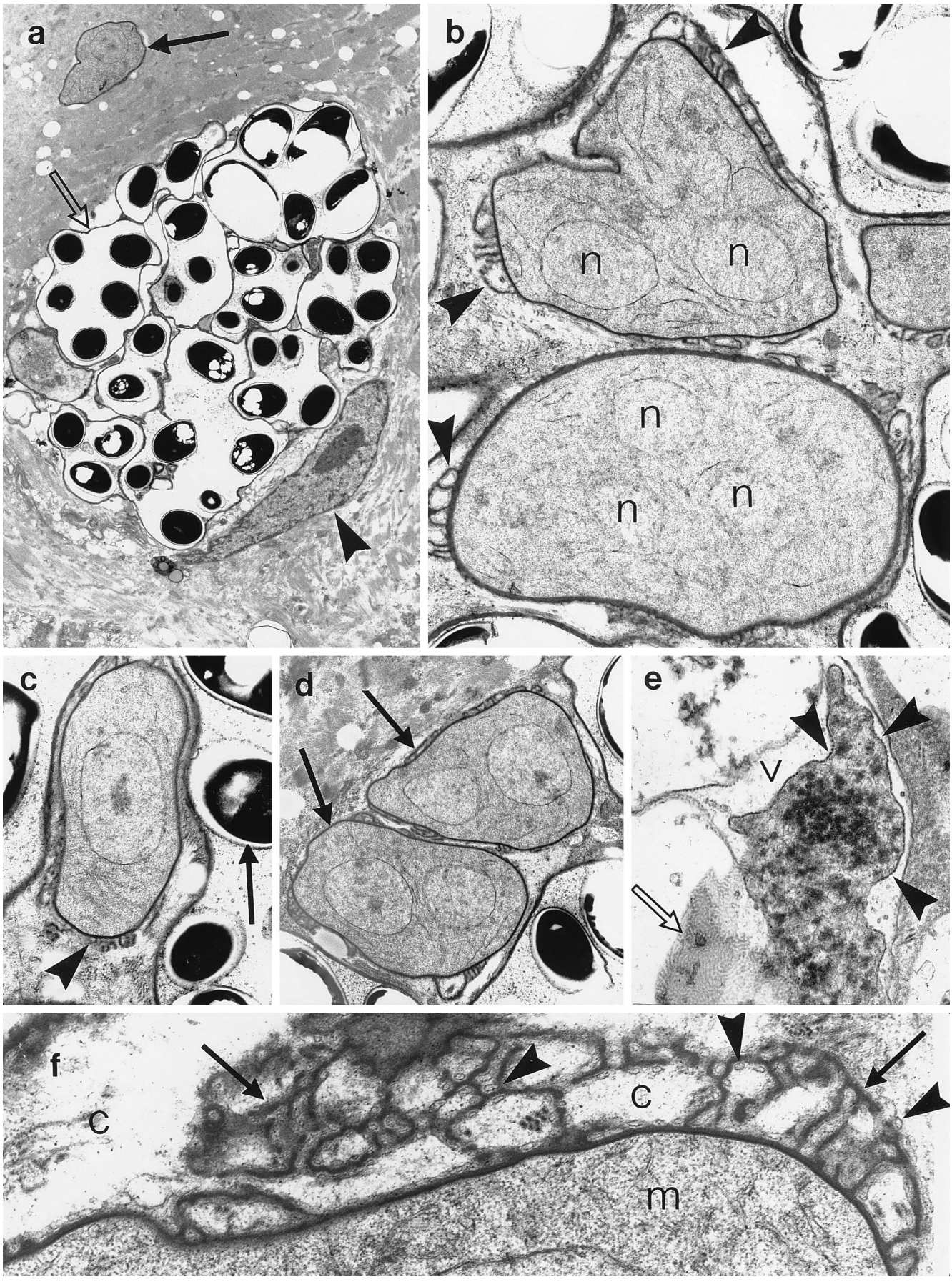

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Routine blood cultures for *M. avium* complex infection taken at admission returned positive 19 days later. Specific therapy was not commenced, as the patient was improving without symptoms suggestive of *M. avium* complex infection. Two months later, the fever recurred without myalgia or increased weakness, and therapy with rifabutin, ethambutol, clarithromycin, and ciprofloxacin was commenced with rapid resolution of the fever. He died of progressive HIV disease 5 months after admission with no clinical recurrence of myositis. Permission for postmortem examination could not be obtained.

MATERIALS AND METHODS

A conjunctival smear was stained with the modified trichrome stain (32). Urine, feces, and nasal washings were cultured for bacteria, fungi, and mycobacteria by standard methods and were also examined for microsporidia with the fluorescent Uvitex 2B stain (31). The muscle biopsy specimens were not processed for routine microbial culture. However, they were sent directly to a reference laboratory for microsporidial culture as described elsewhere (22).

The skeletal muscle biopsy was fixed in phosphate-buffered formalin, processed to a paraffin block, sectioned, and stained with hematoxylin and eosin (H and E), periodic acid-Schiff reagent after diastase digestion, Warthin-Starry stain, Masson's trichrome, Gram's (Brown Brenn) stain, methenamine silver, and Giemsa stain.

For electron microscopy, biopsies were fixed in 2.5% glutaraldehyde, buffered in 0.1 M sodium cacodylate, at room temperature overnight. Further fixation for 60 min in 1% osmium tetroxide and 0.2 M sodium cacodylate buffer and block staining in uranyl acetate (3.5%) were followed by washing and graded dehydration in ethanol and then acetone before embedding in Spurr's resin. Staining was carried out in uranyl acetate (20 min) and Reynold's lead citrate (7 min). Sections were also cut for toluidine blue staining for light microscopy examination, and multiple sections from several different blocks were subsequently viewed with a Philips 410 electron microscope.

RESULTS

Microbiology. *M. avium* was isolated from blood cultures taken at admission, and *Campylobacter jejuni* was grown from a fecal specimen taken 35 days after admission. Methicillin-resistant *Staphylococcus aureus* was isolated in sputum taken 60 days after admission. No other cultures revealed any significant pathogens.

Light microscopy. Microsporidia were detected in nasopharyngeal washings. Multiple specimens of urine and feces were repeatedly negative for microsporidia.

A conjunctival smear stained with the modified trichrome stain showed that many of the epithelial cells contained large aggregates of reddish-orange, oval spores with the suggestion of a surrounding vacuole, the sporophorous vesicle (Fig. 1a). Similar extracellular spores 3 to 5 μm long were present in the background. The free spores were characterized by an ovoid to slightly irregular shape, a mildly birefringent exospore, and an oval region of pallor, the posterior vacuole, towards one pole. The posterior vacuole indented a darker red band towards the opposite anterior pole, probably representing the sporoplasm and the polaroplast, and a red dot present at the anterior pole was consistent with the anchoring disc of the polar tube.

Paraffin sections of skeletal muscle revealed discrete lesions consisting of a central zone of fibrosis and degenerate skeletal muscle myofibers, surrounded by myofibers of approximately normal size containing a variable number of abutting polygonal sporophorous vesicles (Fig. 1b and c). These vesicles had a discernible eosinophilic wall and contained up to 32 spores or spore precursors each up to 5 μm long (Fig. 1e and g). These stained with the H and E, Giemsa, and Gram stains and stained particularly clearly with the Warthin-Starry stain (Fig. 1d and f). In outlying myofibers, where only one or two sporophorous vesicles were revealed by the H and E stain, the Warthin-Starry stain demonstrated the presence of single meronts resting in myofibers (Fig. 1d). Closer to the center of the lesions, the sarcoplasm of the myofibers either was totally replaced by closely packed sporophorous vesicles with nuclear debris caught between vesicles or showed eosinophilic degeneration (Fig. 1c). There was a minimal inflammatory response composed of macrophages (Fig. 1h). The spores did not stain with mucicarmine or methenamine silver stain.

Electron microscopy. All stages of development of the microsporidian were surrounded by an electron-dense surface coat external to its plasma membrane (Fig. 2a, b, c, d, f, and g). The coat had sprouting, branching, and anastomosing "sunflare"-like projections extending up to 100 nm into the lysed sarcoplasm of the myofiber (Fig. 2b, c, d, and f). Focally, the outer surface of the electron-dense coat appeared to be covered by a membrane but closer examination revealed that these membranes belonged to flattened vesicles probably derived from the host cell cytoplasm (Fig. 2f). Within this coat, the meront, up to 7 μm long, lay in direct contact with the host cell cytoplasm (Fig. 2c). Nuclei were unpaired (unikaryotic). In merogony, division was mainly of binucleate meronts to yield two uninucleate meronts by binary fission (Fig. 2d and 3a), although some multinucleated meronts with four or more nuclei were present (Fig. 2b) and divided by plasmotomy, that is, segmentation into smaller fragments containing a variable number of nuclei, the division continuing until uninucleate meronts were produced. At all stages, the meront was tightly surrounded by the surface coat, which was the last structure to divide, often leaving the daughter meronts attached by the external coat (Fig. 3a).

Following division, up to 12 daughter meronts were seen in one muscle cell. Not all entered sporogony simultaneously, and merogony continued alongside sporogony in the same myofiber (Fig. 3a).

In sporogony, the sporont membrane detached itself from the surface coat, leaving the thick coat as the envelope of the sporophorous vesicle (Fig. 3b). The sporont acquired a new surface coat by deposition of electron-dense material initially as spiralling bands and then as a complete coat by coalescence of these bands (Fig. 2e). The uninucleate sporont then entered into a series of nuclear and cytoplasmic divisions by binary fission. It was not certain that sporonts were always uninucleate at the time of separation from the surface coat, but sporogonic cells within the sporophorous vesicle never had more than two nuclei (Fig. 3b). Within early sporophorous vesicles, there was a finely granular matrix containing tubules, which became more obvious and increased in number in later vesicles (Fig. 3c).

FIG. 2. (a) Multiple sporophorous vesicles closely abutting within a skeletal muscle cell (open arrow) with a single meront (arrow). Note the skeletal muscle nucleus (arrowhead). Magnification, ×1,600. (b) Meronts undergoing division. The upper meront has two definite nuclei (n) and is undergoing probable binary fission, while the lower meront has three possible nuclei, and division will be by plasmotomy. Note the branching, anastomosing, sun flare extensions of the thick outer coat (arrowheads). Magnification, ×4,400. (c) Uninucleate meront. Note the thick outer coat (arrowhead) and spores (arrow) in adjacent sporophorous vesicles. Magnification, ×4,400. (d) Two adjacent binucleate meronts with thick outer coats (arrows). Magnification, ×3,000. (e) Sporonts within a sporophorous vesicle (v), showing the periodic focal thickening of the outer coat of the sporont (arrowheads) also seen in a tangential cut as a "fingerprint" of spiralling thickening (open arrow). Magnification, ×10,400. (f) Branching, anastomosing sun flare extensions (arrows) of the thick outer coat of a meront (m), extending into the lysed cytoplasm (c) of a skeletal muscle cell. Note the flattened vesicles of host-derived membranes (arrowheads) on the surface and within branches. Magnification, ×7,100.

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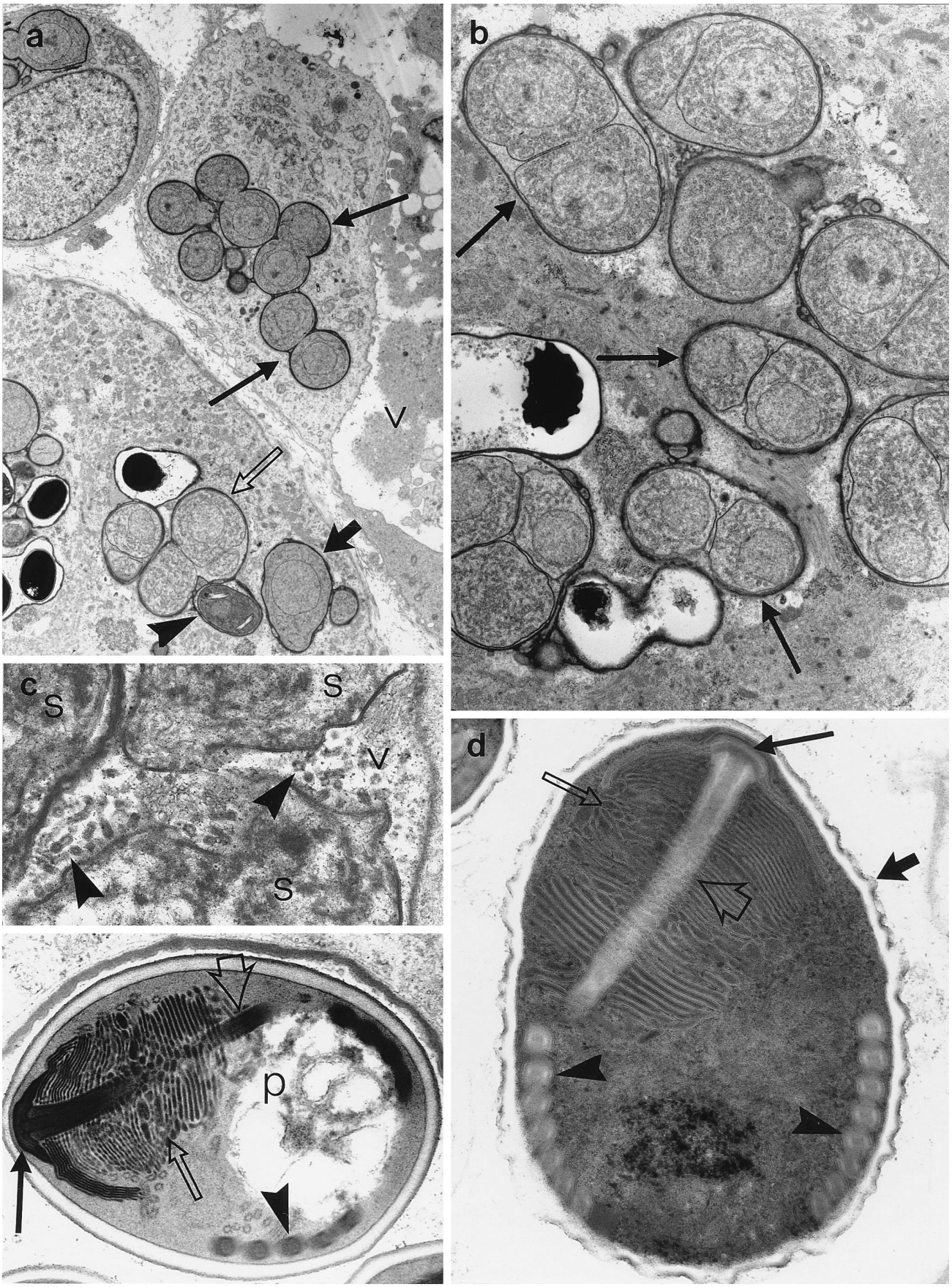

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The final division products of sporogonic divisions were uninucleate sporoblasts (Fig. 3a) which matured into spores. Spores had a distinct polar tube, which was usually arranged as a single layer coiled around the large posterior vacuole (Fig. 3d and e). The number of coils was usually 11, including an occasional coil that was "out of line." The anchoring disc and polar sac from which the straight section of the polar tube extended lay at one pole, often slightly lateral to the center, and the membranes of the polaroplast extended posteriorly from it to surround the straight section of the polar tube. The polaroplast membranes were more tightly packed anteriorly than posteriorly. The endospore was thick, while the exospore was rugose (Fig. 3e).

The sporophorous vesicles appeared to rupture when the membrane of the myofiber ruptured. Macrophages were seen containing sporoblasts and spores in various stages of degeneration and even intact sporophorous vesicles, usually within phagocytic vacuoles. In one case, meronts undergoing division were seen in an endothelial cell of a capillary (Fig. 3a).

DISCUSSION

We have described a case of *Pleistophora* sp.-like microsporidian infection involving skeletal muscle, the conjunctiva, and the nasal sinuses in a human. This is similar to the pattern of dissemination seen with *Encephalitozoon* spp. such as *E.* (*Septata*) *intestinalis* and *E. hellem*, but the prominence of muscle involvement is unique.

The clinical presentation of our patient was similar to that described by Chupp et al. (6). The patient had a previous AIDS-defining illness and an advanced stage of immunodeficiency with a CD4 count of 6×10^6/ml. He presented with a 2-month history of fever with progressive muscle weakness and wasting, and muscle pain was a prominent feature, requiring opiate analgesia. This clinical picture is consistent with a number of HIV-related conditions, including HIV-associated myopathy, zidovudine-induced myopathy, and polymyositis. However, the severity of the symptoms was unusual in our experience and prompted the muscle biopsy.

In contrast to the case of Chupp et al. (6), there was a prompt therapeutic response, highlighting the importance of obtaining a microbiological diagnosis. Albendazole has been used successfully in the treatment of *E. intestinalis* infection (10, 34) and has recently been shown to have in vivo and in vitro activity against the microsporidia *Nosema bombycis* (15) and *E. cuniculi* (7). The initial report of microsporidial myositis of Ledford et al. (24) suggested a clinical response to trimethoprim-sulfamethoxazole followed by sulfadiazine, although this was not confirmed by muscle biopsy. The severity of our patient's illness and the lack of information concerning therapeutic regimens led us to use combination therapy with albendazole, pyrimethamine, and sulfadiazine. The rapidity of the clinical response, with reduction in pain within 24 h and resolution of the muscle contractures in 3 weeks, was striking and was mirrored by the fall in the CK value to normal within 6 weeks. It is not possible to determine the active agent, although folate antagonists did not appear to be effective in the case described by Chupp et al. (6).

The classification of this new microsporidian is of interest. The merogonic stage of the organism is enveloped by a thick surface coat which is probably secreted by the meront, and this coat forms the envelope of the sporophorous vesicle when the sporont separates from its initiating sporogony. This separation creates the internal space of the defining sporophorous vesicle. A variable number of spores is produced within this vesicle. These characteristics are consistent with the genus *Pleistophora*. However, the sporont of the microsporidian we have described undergoes repeated binary fission to produce uninucleate sporoblasts, the sporogonial stages in our material never having more than two nuclei. This is in contrast to the current definition of *Pleistophora* sp., which states that the sporont is a multinucleate plasmodium which divides within the sporophorous vesicle by a series of fissions into smaller and smaller pieces with a variable number of nuclei, until the uninucleate sporoblasts are produced (4, 5). Also, the type species, *P. typicalis*, from the fish *Myoxocephalus scorpius*, lacks the sunflare-like projections we found on the thick outer surface coat, and the thickening of the sporont coat lacks the initial periodic thickening seen in our case (5). Furthermore, an illustration by Cali (1) of material from the *Pleistophora* sp. described by Ledford et al. (24) shows a merogonial plasmodium with many nuclei (six in one plane). The surface coat appears similar, but it is possible that it is a different species.

On this basis, the microsporidian described here should be placed into a new genus, and this has been proposed on the basis of the cultured material (21, 22). Comparison of the 16S rRNA gene sequence using spores derived from in vitro cultures of our patient's muscle biopsy and spores from an in vitro culture of a *Pleistophora* species from fish (which accords with the current definition of the genus) will provide information on the degree of relatedness of these parasites. The large spore size (2.5 by 4 μm, to 3 by 5 μm) provides a clue to the presence of *Pleistophora*-like organisms in light microscopy of clinical specimens which can then be confirmed by electron microscopy. This is an important differentiating characteristic from *Encephalitozoon* sp., which has spores of 2.0 to 2.5 by 1.0 to 1.5 μm (4), and *E. intestinalis*, which has spores of 1.2 by 2 μm (personal observation). Histologically, in tissue sections, the differential diagnosis includes toxoplasmosis, sarcocystis, and leishmaniasis but the distinctive large spores in vacuoles provide a highly significant clue to the diagnosis.

A repeat muscle biopsy was not performed, but microsporidia could not be demonstrated in nasal secretions after 4 weeks of treatment. Long-term suppressive therapy with albendazole was continued after clinical resolution because of the lack of information on the likelihood of relapse with cessation of treatment. However, other protozoal infections in AIDS patients, including *Toxoplasma gondii*, *Isospora belli*, and *Cryptosporidium parvum*, all require suppressive treatment after the initial therapy to prevent relapse, and intuitively it seems likely that such a profoundly immunocompromised patient would have a high probability of relapse.

The mode of acquisition of this *Pleistophora*-like organism is unknown. *Pleistophora* spp. have not been described in Australian terrestrial mammals, although one species, *P. hyphessobryconis*, is a pathogen of neon tetra fish (28a). However,

FIG. 3. (a) Meronts undergoing binary fission (arrows) and remaining attached by their outer coat in an endothelial cell (v, vascular lumen). Sporonts dividing in an adjacent skeletal muscle cell (open arrow) are visible. A sporoblast (arrowhead) and meront (short arrow) are also present. Magnification, ×1,600. (b) Sporonts undergoing division in a skeletal muscle cell (arrows). Magnification, ×3,000. (c) Sporoblasts (s) within a sporophorous vesicle (v). Note the tubules (arrowheads) in the matrix of the vesicle. Magnification, ×7,200. (d) Spore with anchoring disc (arrow), straight portion of polar tube (open short arrow) extending through the polaroplast (open arrow), posterior vacuole (p), and tangential sections through coils of tube (arrowhead). Magnification, ×12,000. (e) Spore with rugose exospore (short arrow), polar tube (open short arrow), anchoring plate (arrow), polaroplast (open arrow), and coils of tube (arrowheads). Magnification, ×21,000.

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ultrastructurally, *P. hyphessobryconis* differs from the species in humans (4). Our patient was born in Australia and had a significant travel history. He kept a dog but otherwise had no contact with animals, including aquarium fish. The route and source of the initial infection and the method of dissemination cannot be determined.

Another Australian group has reported microsporidial sporonts, sporoblasts, and spores in partially digested striated muscle cells in the feces of an HIV-infected patient with diarrhea (25). It was thought that these organisms were *Nosema* sp. cells which had been ingested in food and represented an incidental finding. The microsporidial stages showed apansporoblastic development; that is, they were not enclosed in sporophorous or parasitophorous vacuoles and had diplokaryotic nuclei, establishing them as members of the genus *Nosema*. The presence of *Nosema* sp. organisms in undigested food suggests a possible route of infection for microsporidia. Further, it suggests that finding microsporidia in the feces of AIDS patients may not be diagnostic of infection, reinforcing the need for a diagnostic biopsy.

This report describes a case of severe myositis in an HIV-infected patient associated with a new organism, a microsporidium related to *Pleistophora* spp. The clinical and biochemical response to therapy highlights the importance of obtaining diagnostic tissue. It seems likely that such an infection would arise only in a severely immunosuppressed individual. The diagnosis should be entertained for such patients presenting with clinical myositis and/or conjunctivitis. Light microscopy findings of characteristic spores indicate consideration of antimicrobial therapy. Electron microscopy may identify the particular organism and help determine therapy and prognosis. Specific culture is not readily available, nor are serological tests for diagnosis.

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