agents. *M. abscessus*, in particular, is highly resistant to multiple antimycobacterial drugs. The isolate from our patient was found to be susceptible to amikacin, clarithromycin, cefoxitin, cefmetazole, imipenem, and tobramycin and resistant to trimethoprim-sulfamethoxazole, doxycycline, and ciprofloxacin.

The results of susceptibility studies suggest that amikacin and clarithromycin are the agents most consistently effective against *M. abscessus*. Cefmetazole, cefoxitin, imipenem, and tobramycin may have a role in multiple drug regimens [9]. Monotherapy has proven to be disappointing for the treatment of infections due to *M. abscessus*, and a combination of at least two-to-three active antibiotics is necessary. The optimal duration of therapy has yet to be determined. Previous experience suggests that in disseminated or deep *M. abscessus* infections, antimicrobial treatment should be continued for at least 4–6 weeks after complete resolution; this usually requires 6–12 months of treatment. However, more prolonged courses for years may be necessary in severely immunocompromised patients [10]. It is possible to administer iv therapy for 2–4 months and then switch to oral medications if the organism is susceptible to multiple oral drugs.

Our case illustrates the unusual occurrence of vertebral osteomyelitis due to one of the atypical mycobacteria, *M. abscessus*. Treatment of these infections is difficult and requires a prolonged course of combination antimicrobial agents and aggressive surgical intervention.

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**Pyomyositis Due to Mycobacterium haemophilum in a Patient with Polymyositis and Long-Term Steroid Use**

*Mycobacterium haemophilum* is emerging as a pathogen of immunocompetent children and immunocompromised adults, particularly those who have AIDS, have received organ transplants, and have undergone cytotoxic chemotherapy [1, 2]. Frequently, results of acid-fast stains of aspirates or biopsy specimens are positive, but the organism fails to grow under routine conditions used to isolate mycobacteria [1]. Cases of tenosynovitis, septic arthritis, and osteomyelitis attributed to *M. haemophilum* have been described [1, 2]. To our knowledge, pyomyositis due to *M. haemophilum* has not been reported previously. We describe a patient with multisite pyomyositis due to *M. haemophilum*, which occurred during long-term steroid treatment for polymyositis.

A 60-year-old man was admitted to the hospital in June 1995 because of ulceration over the extremities and dyspnea and fever. Polymyositis was diagnosed 14 months before this admission. Prednisolone (30 mg/d) was given for >1 year. The patient was Taiwanese, lived in an urban area, and raised fish and birds at home; he had no travel history in recent years. The patient had a history of fever, lower extremity tenderness, and erythematous changes in both thighs and the left arm 4 months before this admission, and despite treatment with antibiotics, ulcers developed over these areas 1 month before admission.

On physical examination, the temperature was 38.2°C. Auscultation revealed bilateral crackles on inspiration over the lower lungs. Engorged jugular veins, a deep ulcer over the left arm with exposure of underlying muscle, and a fistula of the muscle layers over the right thigh with purulent drainage were noted. Both thighs and the left arm were swollen, erythematous, and tender to palpation. A chest radiograph showed pulmonary edema and cardiomegaly. The WBC count was 11,900/mm^3^ with 91% neutrophils. Tests for antibodies to HIV were negative. A CT scan of the lower extremities showed low-density lesions in all three compartments of both lower extremities (figure 1), extending from both knees to the thighs and the gluteal region. A three-phase radionuclide bone scan was performed and did not show evidence of osteomyelitis. We describe a patient with polymyositis 14 months before this admission. Prednisolone (30 mg/d) was given for >1 year. The patient was Taiwanese, lived in an urban area, and raised fish and birds at home; he had no travel history in recent years. The patient had a history of fever, lower extremity tenderness, and erythematous changes in both thighs and the left arm 4 months before this admission, and despite treatment with antibiotics, ulcers developed over these areas 1 month before admission.

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Figure 1. Contrast-enhanced CT scan of both thighs of a patient with Mycobacterium haemophilum pyomyositis showing increased muscle volume and diminished muscle density (arrowheads) in all three compartments. A skin defect (arrow) is visible at the medial side of the right thigh.

intermingled with lymphocytes and plasma cells among the fragmented and degenerated muscle fibers. Kinyoun acid-fast stains demonstrated acid-fast bacilli (AFB) at the three sites. Cultures of the biopsy specimens were negative for bacteria and fungi. Antimycobacterial therapy with isoniazid, ethambutol, and rifampin was instituted. The initial cultures of wound discharge and biopsy specimens were positive for mycobacteria after 10 weeks of incubation. Cultures of sputum, bronchial lavage fluid, blood, and bone marrow specimens under routine conditions at 37°C were all negative for mycobacteria.

Because M. haemophilum infection was suspected, subcultures of the initial growth were cultivated on chocolate agar and a standard Lowenstein-Jensen slant at 30°C. The therapeutic regimen was changed to oral ethambutol, rifampin, and clarithromycin and iv ciprofloxacin and amikacin. The wounds did not resolve; consequently, repeated debridement of the right thigh was performed, and the biopsy specimen from that site still yielded AFB. The patient died of fungemia due to Candida glabrata 3 months after admission.

The subculture yielded good growth on chocolate agar after 3 weeks of incubation but was negative on a Lowenstein-Jensen slant, even after 12 weeks of incubation. The organism, a nonchromogen, was negative for niacin accumulates, nitrate reduction, urease activity, catalase reaction at 68°C, and tellurite reduction [3]. A fatty-acid profile generated by gas-liquid chromatography was compatible with that of M. haemophilum, as described previously [4]. MICs for the isolate, obtained by using the Etest (PDM Epsilometer; AB BIODISK, Solna, Sweden) [5] on chocolate agar after a 3-week incubation at 30°C, were 0.25 μg/mL for rifampin, 0.125 μg/mL for clarithromycin, and 0.5 μg/mL for ciprofloxacin.

Skeletal muscle is believed to be highly resistant to infection [6]. Although the occurrence of pyomyositis remains rare, it is increasingly recognized in immunocompromised patients, especially those with AIDS [6]. Mycobacterial infection of skeletal muscle is very rare; large muscles are involved more frequently, and the condition usually presents as localized muscle involvement by direct extension from a proximate focus of infection [7]. Most of the reported cases of mycobacterial myositis have been caused by Mycobacterium tuberculosis. However, persons with defects in immunity are more susceptible to those organisms encountered less frequently. Necrotizing pyomyositis caused by Mycobacterium avium complex (MAC) has been described in patients with AIDS [6]. Our patient developed M. haemophilum pyomyositis in multiple areas after >1 year of steroid therapy for polymyositis. It is well known that glucocorticoids suppress the activation of T cells, the function of natural killer cells, and the amplification of cell-mediated immunity. Nuñez et al. [8] described a patient with polymyositis who developed disseminated MAC infection during treatment with prednisolone. M. haemophilum infection has been described in patients with a history of long-term steroid use for rheumatoid arthritis [2]. Susceptibility to pyomyositis is increased by muscle damage from local mechanical trauma [6]. We do not know whether the muscle damage from pyomyositis predisposed our patient to muscle involvement by M. haemophilum infection.

Because M. haemophilum requires iron-enriched media and an optimal temperature between 30°C and 32°C for growth, the diagnosis of infection due to this organism can be missed or delayed [1, 2]. In the present report, it is likely that the initial growth occurred on routine mycobacterial media because the heme from the bloody biopsy specimen met the organism’s nutritional requirements. However, growth was slow because of incubation at 37°C. According to published reports, lesions and symptoms of M. haemophilum infection regressed in two-thirds of patients after treatment [1, 2]. Enhanced immune function and a combination of ciprofloxacin, clarithromycin, and one of the rifamycins appear to be effective for treatment of M. haemophilum infection [2]. Surgi-
cal drainage with debridement of necrotic tissue may be mandatory when extensive pyomyositis occurs.

In conclusion, a high index of suspicion and selection of the proper culture conditions should help clinicians identify cases of pyomyositis due to *M. haemophilum* and initiate appropriate treatment.

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**References**


**Detection of Human Papillomavirus Type 35 in a Nodular Cutaneous Tumor in a Patient Infected with Human Immunodeficiency Virus**

Numerous studies of HIV-seropositive and HIV-seronegative women and homosexual men have shown an increased incidence of human papillomavirus (HPV) infection and HPV-related neoplasia in HIV-positive individuals [1]. The neoplasms described in HIV-infected patients include basal cell carcinoma, squamous cell carcinoma, Bowenoid papulosis, Bowen’s disease, cloacogenic carcinoma, and malignant melanoma [2]. The degree of immunodeficiency (CD4 cell counts) is an important factor with respect to the prevalence of HPV-associated dysplastic and neoplastic lesions. The relative risk for invasive anal cancer among homosexual men, compared with the general population, increases significantly, especially after the diagnosis of AIDS [1]. High-risk HPV types 16 and 18 are detected most frequently, but other HPV types are also found in high-grade squamous intraepithelial lesions (SILs) in patients with AIDS [1]. Because of their immunocompromised status, HIV-infected patients are less able to suppress cutaneous premalignant and malignant diseases; therefore, histological examination of any suspicious lesion has been suggested for these patients [2].

We describe an HIV-infected patient with markedly impaired cellular immunity (1996 CD4 cell count, 40–70/μL), but no other evidence of AIDS-defining disease. A 49-year-old male had had deep dermal nodules of the scrotal skin since January 1996 (figure 1). In May 1996, histopathologic examination of a scrotal biopsy specimen was performed. The epidermis showed irregular acanthosis with many enlarged and hyperchromatic nuclei, as well as dyskeratotic and vacuolated keratinocytes. Mitoses could be detected above the basal layer. A second biopsy specimen examined in November 1996 again displayed pronounced acanthosis and papillomatosis with mitoses present above the basal layer. Koilocytic cells could be detected in the upper portions of the epidermis, and many nuclei were hyperchromatic and irregular, sometimes with prominent nucleoli.

By use of PCR with consensus primers MY09/MY11 [3], HPV DNA was detected both in a scrotal-skin swab specimen obtained in January 1996 and in the biopsy specimen from May 1996. By restriction fragment length polymorphism (RFLP) analysis of the PCR product and subsequent hybridization with a generic oligonucleotide probe [4], HPV 35 was identified in both specimens. The presence of HPV 35 was confirmed by sequence analysis of the PCR product. In November 1996, the nodular tumor lesions were excised and the patient subsequently received treatment with IFN. Up to 6 months after surgery, no recurrence of symptoms was observed.

The clinical manifestations of the scrotal skin lesions described herein are unusual for HPV-associated anogenital lesions. Typical morphological features of condyloma acuminatum, SILs, carcinoma in situ (Bowenoid papulosis or Bowen’s disease), or invasive carcinoma were not detectable by histopathologic evaluation, although the dysplastic epithelial changes somewhat resembled bowenoid papulosis. However, the koilocytic epithelial changes indicated etiologic involvement of HPV infection.

Among carcinomas in situ (Bowenoid papulosis or Bowen’s disease) and cancers of the anogenital tract, HPV 16 is the HPV type detected most frequently [5]. HPV 35 was first detected in a cervical adenocarcinoma [6]. Later, it was detected in other sites of the lower anogenital tract (i.e., vulva, vagina, penis, and anus). HPV 35 has also been identified in Bowenoid dysplasia of the perianal area [7]. HP 35 had been commonly referred to as an HPV type with intermediate risk for tumor induction that is most prevalent in high-grade SILs [8]; however, due to its detection in cervical and laryngeal carcinomas, HPV 35 is now considered...