Cutaneous Vascular Lesions and Disseminated Cat-Scratch Disease in Patients with the Acquired Immunodeficiency Syndrome (AIDS) and AIDS-Related Complex

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Cutaneous lesions develop frequently in patients infected with human immunodeficiency virus (HIV). We describe the clinical features of four patients with the acquired immunodeficiency syndrome (AIDS) or AIDS-related complex who developed angiomatous nodules involving skin and bone, 2 of whom were scratched by a cat. Some of these lesions were clinically indistinguishable from Kaposi sarcoma. When examined with Warthin-Starry staining and electron microscopy, these nodules were noted to contain numerous clumps of a bacterium. Immunoperoxidase staining with an antiserum raised against the cat-scratch disease bacillus stained these organisms in all patients. Cat-scratch disease is usually a self-limited infection, but complicated or prolonged infections have been described in both normal and immunocompromised hosts. In our patients infected with HIV, manifestations of systemic cat-scratch disease included angiomatous nodules, severe systemic symptoms of fever, chills, night sweats and weight loss, elevated erythrocyte sedimentation rate, and decreased hematocrit. Cutaneous lesions involved the face, trunk, and extremities and numbered 2 to greater than 60; osseous lesions involved the fibula, radius, femur, and tibia, and were present in two of four patients. Treatment with x-ray therapy, intraleisional vinblastine, penicillin, doxycycline, or antitymocobacterial antibiotics resulted in complete and rapid resolution of the cutaneous and osseous lesions, and the accompanying signs and symptoms of systemic infection. In patients with AIDS or AIDS-related complex, angiomatous nodules should be carefully evaluated for the presence of this organism, which can be treated and cured with antibiotic agents.


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Cutaneous vascular lesions in patients infected with human immunodeficiency virus (HIV) are usually considered to be Kaposi sarcoma. In 1983, however, a patient with the acquired immunodeficiency syndrome (AIDS) developed angiomatous nodules in which bacteria were found (1). This initial report, and other subsequent reports (2, 3) describing histiocytoid and epithelioid hemangiomas in persons infected with HIV suggest that such nonmalignant angiomatous lesions are not rare. We discuss the clinical features of four patients with AIDS and AIDS-related complex with angiomatous cutaneous and osseous lesions and systemic symptoms. These cutaneous vascular lesions contain a bacterium that appears to be identical to the organism that causes cat-scratch disease (4, 5). We provide the first description of the natural history of this infection associated with the cat-scratch bacillus, and its response to antimicrobial therapy.

Patients and Methods

Patient 1

A 43-year-old homosexual man from Acapulco presented in February 1987 with a right wrist mass of approximately 4 months' duration, and fever and chills of 2 weeks' duration. He was scratched by a cat in November 1986. A painful, 5-cm × 3-cm firm, non-pulsatile right wrist mass was noted, without adenopathy (Figure 1, left). A radiograph of the right wrist showed a bony defect in the distal radius, and computed tomography showed a destructive mass arising from the distal volar radius consistent with tumor, especially a sarcoma (Figure 1, right). A bone scan showed focal, intense uptake with a central avascular or necrotic component.

In March 1987 the patient was noted to have oral thrush and chronic perirectal herpes simplex infection. Additional findings included positive results on an HIV antibody test, a normal chest radiograph, an erythrocyte sedimentation rate of 58 mm/h, a leukocyte count 2.5 × 10⁹/L, and a hematocrit of 0.35. A VDRL was reactive at the 1:128 dilution; the patient stated that he had been treated for secondary syphilis with three penicillin injections 1 year before admission, and denied subsequent exposure. Open biopsy showed the mass to be a firm, fibrous, destructive lesion causing frank erosion of the cortical bone. Pathologic findings showed fragments of fibrous connective tissue, with bone and chronic inflammation. The Steiner stain for treponemes was negative.

In June 1987 the mass began to enlarge, and new nodules appeared deep in the right thigh, on the upper right suprascapular region, and above the left, right, and right anterolateral thigh. The leukocyte count was 4.6 × 10⁹/L; erythrocyte sedimentation...
rate, 92 mm/h; hematocrit, 0.35; and VDRL, positive at 1:32.

The patient was treated for gummatous syphilis with benzathine penicillin, 2.4 million units, intramuscularly in July 1987. An initial decrease in symptoms and size of the right wrist lesion occurred. However, despite continuing penicillin injections, the right wrist lesion began to grow larger and more painful, and disseminated to new sites. The original lesion became extremely painful and continued to grow rapidly until it was a circumferential, erythematous mass that completely limited flexion and extension of the wrist. A radiograph of the right wrist showed increased cortical bone loss and periostitis, in addition to a marked increase in the soft tissue mass.

In August 1987 antibiotic therapy with oral erythromycin, 500 mg every 4 hours, was started. Within 2 days, draining sinus tracts opened in the right thigh lesion, and a fluctuant pus-filled area developed within the wrist lesion. A Gram stain of aspirated pus showed many polymorphonuclear cells without organisms; atypical, slightly acid-fast organisms were noted after Kinyoun staining, but mycobacterial, fungal, viral, and bacterial cultures were negative. Two weeks after beginning erythromycin therapy, the wrist and thigh lesions had markedly decreased in size, but continued to drain. The leukocyte count was 3.1 \times 10^9/L; erythrocyte sedimentation rate, 118 mm/h; hematocrit, 0.30; and a radiograph of the right wrist showed improvement. The lesions and symptoms continued to resolve over the next 4 weeks of treatment.

By December 1987 the wrist, thigh, and eyebrow lesions had completely resolved (Figure 1, middle). The leukocyte count was 2.3 \times 10^9/L; erythrocyte sedimentation rate, 52 mm/h; and hematocrit, 0.38. A bone scan showed continued intense uptake of technetium in the right wrist, but a gallium scan of the same region showed no uptake, and these scans were considered to be consistent with an osteoblastic reaction, without evidence of osteomyelitis. Other regions of previous gallium uptake in June 1987 also had resolved. On follow-up in March 1988, the patient had regained lost weight, returned to work and his usual weightlifting exercise, and felt well without significant medical problems, fevers, chills, or skin lesions; the leukocyte count was 2.6 \times 10^9/L, and the hematocrit 0.38.

Patient 2

A 52-year-old white man, HIV-antibody positive, noted onset of fever and fatigue in January 1986. In April 1986 he developed a red skin lesion on the chest, and subsequently developed similar lesions on the right temple, trunk, and extremities. A shave biopsy of the temple lesion was followed by regrowth of the lesion 4 days later. In July 1986 the patient reported fever, chills, night sweats, and a 7-kg weight loss. The patient had an absolute T4 subset count of 7; a leukocyte count of 5.1 \times 10^9/L; and an erythrocyte sedimentation rate of 53 mm/h.

In September 1986 several typical lesions of psoriasis on the right lower extremity, and 60 erythematous, exophytic, angiomatous lesions measuring 0.5 cm to 2 cm on the face, back, chest, arms, and legs were seen. The largest lesion measured 4 cm in diameter and projected 4 cm off the chest wall; the patient received two radiation treatments to this lesion (400 rad total). Another lesion was injected once with vinblastine, but neither of these treatments had any effect.

The patient was hospitalized in October 1986 because of shortness of breath and cough; oral thrush and hairy leukoplakia were present at this time. Interstitial infiltrates were seen throughout both lung fields, but bronchoalveolar washings and sputum were negative for Pneumocystis carinii. The washings eventually
grew group III, atypical mycobacteria, and the patient began isoniazid and rifampin therapy. Shortly after beginning antibiotic therapy in November 1986, the skin lesions began to resolve, and eventually, all resolved completely. No other therapy was given, nor did the lesions recur. Anemia and liver dysfunction progressed in February and March 1987, and the patient died in April 1987 of hepatic failure.

Patient 3

A 51-year-old homosexual white man presented in October 1985 with neutropenia, thrombocytopenia, and mild fatigue of 6 months' duration. At this time of initial presentation, his leukocyte count was 3.1 x 10^9/L; erythrocyte sedimentation rate, 15 mm/h; VDRL, nonreactive; and chest radiograph, normal. In January 1986 several lesions of molluscum contagiosum were noted on the face. Leukocyte count was 2.9 x 10^9/L; erythrocyte sedimentation rate, 17 mm/h; hematocrit, 0.40; and platelet count 114 x 10^9/L. In October 1986 persistent cough developed, and a chest radiograph showed a new, patchy right upper lobe infiltrate; sputum induction was negative for P. carinii, acid fast bacilli, and fungi. The presentation, his leukocyte count was 3.1 x 10^9/L; erythrocyte sedimentation rate, 32 mm/h; hematocrit was 0.40; and platelet count was 45 x 10^9/L. The patient was anergic.

In August 1987, the patient noted the sudden appearance of a fleshy, raised lesion on the dorsal surface of the left third finger (Figure 2). The lesion grew rapidly, and developed a 1-cm erythematous, cellulitic appearance base, without drainage or fluctuance. Treatment with cephradine, 2 g/d, was started, but was changed to dicloxacillin, 1 g/d, several days later due to drug intolerance, and continued for a total course of 10 days. There was no improvement of the initial lesion, and an additional lesion, a 0.8-cm red-violet, glossy-domed papule (Figure 2), developed in the left inguinal region. At this time, the patient also reported frequent fevers to 39.5 °C, and a persistent cough. The absolute T4 subset count was 54; leukocyte count, 2.2 x 10^9/L; erythrocyte sedimentation rate, 48 mm/h; hematocrit, 0.40; and platelet count 100 x 10^9/L. In September 1987 both lesions were removed by shave biopsy (see Results).

In October 1987 the patient was seen again because of persistent cough. He had a persistent, red, raised lesion on the left third finger; the groin lesion had not recurred. The patient was given doxycycline, 100 mg twice a day, for 14 days for presumed bronchitis. The symptoms and chest radiograph improved. The symptoms and chest radiograph improved. The symptoms and chest radiograph improved.

During this admission, several pigmented skin nodules by 1 week of intravenous pentamidine because of presumed trimethoprim/sulfamethoxazole side effects. A repeat bronchoalveolar lavage was negative for P. carinii, although cytomegalovirus was eventually cultured. During his 1-month hospitalization, he was anergic, and had positive results on fluorescent treponemal antibody test, a reactive VDRL at a 1:2 dilution, and repeatedly negative blood cultures. He received two weekly intramuscular injections of 2.4 mIU benzathine penicillin. At discharge, the T helper to suppressor ratio was 0.1; leukocyte count, 2.2 x 10^9/L; erythrocyte sedimentation rate, 112 mm/h; hematocrit, 0.28; platelet count, 175 x 10^9/L; serum creatinine level, 133 μmol/L; and albumin level, 30 g/L. After discharge, the patient had marked improvement in his respiratory symptoms; a repeat VDRL was positive at a 1:2 dilution, and a urine culture grew cytomegalovirus.

In April 1983 the patient complained of generalized arthralgias and painful swelling in the lower extremities, without antecedent trauma, and was noted to have bilateral lower extremity edema with an area of possible cellulitis. He was treated with dicloxacillin, 1 g/d, for 10 days, without improvement, and was subsequently admitted for intravenous nafcillin therapy. During this admission, several pigmented skin nodules were seen and biopsies were done (see Results). The leukocyte count was 2.9 x 10^9/L; erythrocyte sedimentation rate had risen to 136 mm/h; hematocrit was 0.31; VDRL had increased to 1:8; the antinuclear antibody and rheumatoid factor were negative; and the chest radiograph improved. The symptoms and skin lesions did not improve with nafcillin therapy, and he was discharged on indomethacin therapy.

In May 1983 a bone scan was done because of continued bone pain associated with the skin lesions.
There was abnormal uptake in the left tibia, right fibula, and right radius; this scan was felt to be most consistent with metastatic disease. There were no osseous abnormalities seen on plain films of the extremities. Repeat biopsies of the skin lesions were done. An intramuscular injection of benzathine penicillin, 2.4 mlU, was administered.

Over the subsequent month, the lesions continued to increase in number, with a distribution involving the face, and upper and lower extremities. Lesions also recurred at several sites of previous biopsies. There was pain associated with some of the lesions, some became pedunculated, and trauma occasionally resulted in bleeding for up to 30 minutes. A prominent, 2-cm subcutaneous mass developed in the left preordial area, in addition to a cluster of nodular lesions over the right fibula; there was severe bone tenderness underlying these lesions. A repeat bone scan done 1 month after the initial scan continued to show increased uptake in the proximal anterior left tibia, distal right fibula, and proximal and distal right forearm. The leukocyte count was 3.1 \times 10^9/L; erythrocyte sedimentation rate, 137 mm/h; hematocrit, 0.29; platelet count, 171 \times 10^9/L; and VDRL, positive at 1:4 dilution.

Gastrointestinal symptoms prompted an admission in July 1983, and workup during this hospitalization documented disseminated infection with *Mycobacterium avium-intracellulare*. Antimycobacterial therapy with isoniazid, rifampin, and ethambutol was begun, and within 1 week of institution of therapy, the skin and bone lesions began to improve, with marked diminution in both size and vascularity 2 weeks later. Leukocyte count was 5.2 \times 10^9/L; hematocrit, 0.26; platelet count, 149 \times 10^9/L; serum creatinine level, 194 \mu mol/L; albumin level, 15 g/L; alkaline phosphatase level, 4.9 \mu kat/L; and severe proteinuria was present. He was discharged on ketoconazole therapy for invasive esophageal candidiasis, in addition to antimycobacterial therapy.

In August 1983 clofazimine was added to the regimen. Rapid involution of the lesions continued over the next 2 months, and by September 1983, the facial lesions had cleared completely, with only scattered, hyperpigmented macules remaining at the skin sites previously affected with the pedunculated, nodular lesions. A follow-up bone scan in August 1983 showed no uptake in the previously abnormal right forearm; the right distal fibula and left anterior tibia showed very small, subtle areas of increased radioactive uptake that were markedly decreased compared with the previous bone scan before therapy. The patient returned to work, but had progressive fatigue and wasting in October and November 1983. He died at home in November 1983, of nephrotic syndrome, without recurrence of skin lesions.

**Results**

Skin biopsies from all four patients showed similar features. The angiomatous nodules showed a protuberant contour, often with ulcerated surfaces. Lobular vascular proliferations were present in the subjacent dermis, associated with edematous stroma, surrounded by epithelial collarettes. The endothelial cells lining the vascular spaces were large and protruded into rounded vessel lumina; the cells had vesicular nuclei and central, prominent nucleolus, superficially resembling histiocytes. Mitotic figures, some of which were atypical, were seen. Neutrophils and leukocytoclastic debris were scattered throughout the lesions. The centers of the vascular lobules were necrotic in two biopsies from one of the patients. In the subendothelial areas granular, slightly basophilic deposits were seen at medium magnification (×100). In plane section, these deposits were two to three times larger than the adjacent proliferating endothelial cells (Figure 3, *top*). These deposits stained dark brown to black in a granular pattern with Warthin-Starry stain, and corresponded to clumps of bacteria on electron microscopy. Other stains for organisms including Fite, Steiner, and Brown-Brenn were negative.

The electron microscopic and immunohistochemical findings are fully described elsewhere (4). The electron micrographs showed numerous bacilli, most of which were located extracellularly, within the loose connective tissue surrounding blood vessels (Figure 3, *bottom*). The organisms in all four patients were stained with immunoperoxidase using antisera raised against the cat-scratch disease bacillus (5).

**Discussion**

Cat-scratch disease most frequently occurs in childhood (6). In the normal host, a primary lesion appears at the inoculation site 3 to 5 days after exposure to a cat. Within 1 to 2 weeks regional, usually solitary, lymphadenopathy occurs, which persists for weeks to months. Lymphadenopathy resolves with or without suppuration. Systemic symptoms are usually mild and nonspecific. Some cases of Parinaud oculoglandular syndrome are an unusual form of cat-scratch disease, in which the conjunctiva is the inoculation site (7).

The cause of cat-scratch disease was unknown until Wear and associates (8) discovered a small gram-negative bacterium, identified by Warthin-Starry stain, in 34 of 39 lymph nodes of patients with cat-scratch disease. Since that initial report, Wear and coworkers have found this organism in hundreds of lymph nodes from patients with cat-scratch disease and in primary eye and skin lesions (7, 9). Other investigators have confirmed these findings (10). Sera from patients with known cat-scratch disease react with this bacterium in tissue sections (8). Recently a bacterium has been successfully cultured from lymph nodes of patients with cat-scratch disease and an antisera against this presumed agent of cat-scratch disease has been produced (5).

In 1% to 2% of patients, severe or systemic cat-scratch disease occurs. Although 87% of all patients with cat-scratch disease are under age 18, adults are disproportionately represented among patients with severe disease (11). Most patients with severe disease have persistent, prolonged, or severe symptoms (11).
Rarely, extralymphocutaneous disease has been reported: central nervous system involvement, usually encephalopathy, in more than 40 patients (12); lytic bone lesions in 7 patients (13-15); hepatitis in 4 (16, 17); pulmonary lesions in 2 (16, 18); and hemolytic anemia in 1 (19).

The natural history of cat-scratch disease in the immunocompromised host is unknown. One renal allograft recipient suffered life-threatening sepsis (18). In 1983 an unusual infection was first reported in a patient with AIDS: several, firm, nontender, 2- to 6-cm subcutaneous nodules appeared on the head and extremities (1). Biopsies showed proliferation of blood vessels, with infiltrates of polymorphonuclear leukocytes and histiocytes. Although cultures were negative, small bacilli were identified with the Warthin-Starry stain and by electron microscopy. All lesions healed with erythromycin therapy. In retrospect, immunoperoxidase staining showed that these organisms were cat-scratch disease bacilli (4).

The clinical manifestations of disseminated cat-scratch disease in our series of four HIV-infected patients included systemic symptoms and signs, and cutaneous and osseous lesions (Table 1), with a wide range in the severity of systemic symptoms and number of cutaneous lesions. Patients 1, 2, and 3 presented with systemic symptoms, which improved after antibiotic therapy. In Patient 1, severe systemic symptoms of fever, chills, night sweats, and weight loss began when the right wrist lesion appeared, and resolved completely after 4 months of erythromycin treatment. The hematocrit decreased, and erythrocyte sedimentation rate increased at the time when lesions were most severe, and returned toward normal after treatment and resolution of lesions. No other systemic disease has been diagnosed, and the patient remains well 4
Table 1. Characteristics of Patients with Human Immunodeficiency Virus and Disseminated Cat-Scratch Disease

<table>
<thead>
<tr>
<th></th>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
<th>Patient 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yrs</td>
<td>43</td>
<td>52</td>
<td>51</td>
<td>39</td>
</tr>
<tr>
<td>Cat-scratch exposure</td>
<td>Yes</td>
<td>Unknown</td>
<td>Yes</td>
<td>Unknown</td>
</tr>
<tr>
<td>Cat-scratch disease symptoms</td>
<td>Fevers, chills, night sweats, weight loss, sever bone pain</td>
<td>Bleeding, pain, weight loss, edema</td>
<td>Bleeding, pain, fevers</td>
<td>Bleeding, pain, edema</td>
</tr>
<tr>
<td>Lesions, type (n)</td>
<td>Skin (5); bone (2)</td>
<td>Skin (more than 60)</td>
<td>Skin (2)</td>
<td>Skin (more than 60); bone (3)</td>
</tr>
<tr>
<td>Location</td>
<td>Back, right wrist and thigh, right radius and femur</td>
<td>Face, trunk, extremities</td>
<td>Left third finger, left groin</td>
<td>Face, trunk, extremities, right fibula, radius, and left tibia</td>
</tr>
<tr>
<td>Antibiotic treatment</td>
<td>Penicillin (none); erythromycin (resolution)</td>
<td>Intraloesional vinblastine (none); x-ray therapy (none); isoniazid/rifampin (resolution)</td>
<td>Cephradine (none); doxycycline (none); isoniazid/rifampin (resolution)</td>
<td>Penicillin (none); nafcillin (none); dicloxacillin (none); doxycycline (resolution)</td>
</tr>
<tr>
<td>Associated conditions</td>
<td>Herpes, positive VDRL, oral thrush, neutropenia</td>
<td>Amebiasis, psoriasis, neutropenia, Mycobacterium avium-intracellulare</td>
<td>Neutropenia, thrombocytopenia</td>
<td>Pneumocystis carinii pneumonia, positive VDRL, amebiasis, Candida esophagitis,</td>
</tr>
<tr>
<td>Outcome</td>
<td>Alive; asymptomatic</td>
<td>Dead</td>
<td>Alive; asymptomatic</td>
<td>Dead</td>
</tr>
</tbody>
</table>

* Patients 1, 2, 3, and 4 correspond to same patients numbered 5, 6, 7, and 4, respectively, in reference 4.

It can be difficult or impossible to clinically distinguish exophytic nodules of Kaposi sarcoma from the vascular lesions due to the cat-scratch disease bacillus. Certain clinical and histologic features can facilitate this differentiation (Table 2). The nodules of cat-scratch disease are more friable, and tend to bleed with minor trauma. Although bleeding can also occur with Kaposi sarcoma, it is less common, possibly because many of the vascular spaces in nodular Kaposi sarcoma are slits formed by malignant endothelial cells, not functional vessels. Erythema surrounding the base of the nodule is common in cat-scratch disease lesions, and may initially be mistaken for cellulitis; this erythema is unusual in Kaposi sarcoma. Pain and tenderness are often prominent in cat-scratch disease lesions, and are

Table 2. Clinical and Histologic Features Distinguishing Cat-Scratch Disease Nodules from Kaposi Sarcoma in Patients with the Acquired Immunodeficiency Syndrome (AIDS) and AIDS-Related Complex

<table>
<thead>
<tr>
<th>Features</th>
<th>Angiomatous Nodules of Cat-Scratch Disease</th>
<th>Kaposi Sarcoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical</td>
<td>Friability</td>
<td>Marked</td>
</tr>
<tr>
<td></td>
<td>Erythematous base</td>
<td>Common</td>
</tr>
<tr>
<td></td>
<td>Pain and tenderness</td>
<td>Common</td>
</tr>
<tr>
<td></td>
<td>Presence of osseous lesions</td>
<td>Common</td>
</tr>
<tr>
<td></td>
<td>Response to antibiotic therapy</td>
<td>Complete</td>
</tr>
<tr>
<td>Histologic</td>
<td>Endothelial cells</td>
<td>Polygonal, histiocytoid</td>
</tr>
<tr>
<td></td>
<td>Vascular lumina</td>
<td>Rounded</td>
</tr>
<tr>
<td></td>
<td>Neutrophil clusters</td>
<td>Present</td>
</tr>
<tr>
<td></td>
<td>Basophilic granular material</td>
<td>Present</td>
</tr>
</tbody>
</table>
uncommon in Kaposi sarcoma. In AIDS-associated Kaposi sarcoma, osseous lesions are very rare, whereas such lesions occurred in two of our four patients. Certain histologic features, such as mitoses and atypia of the endothelial cells, may also mislead the pathologist to a diagnosis of malignancy.

Successful treatment of the cutaneous and osseous lesions in our patients was achieved with erythromycin, doxycycline, and the antimycobacterial drugs isoniazid and rifampin. The lesions in our patients were not responsive to penicillin, nafcillin, dicloxacillin, or cephradine. Patient 1 initially received penicillin, and was observed closely during and after weekly administrations. His lesions worsened, and new lesions developed. In contrast, the response of this patient’s lesions to erythromycin was dramatic, with the development of draining sinus tracts within 48 hours, and subsequent rapid resolution of all lesions. The lesions unexpectedly resolved in the other three patients after treatment with isoniazid and rifampin or doxycycline for other conditions. These lesions responded to antibiotic agents that inhibit protein and RNA synthesis, but did not respond to antibiotic agents that inhibit cell wall synthesis. This pattern of sensitivity would be expected for the cell-wall deficient variant of the cat-scratch bacillus isolated by English and colleagues (5).

We believe that the bacterium we have identified in the angiomatous lesions of patients with AIDS and AIDS-related complex is the cat-scratch bacillus. The two living patients who could be interviewed both gave histories of cut scratches. Infection of the conjunctiva in normal hosts with the cat-scratch bacillus produces vascular lesions that have clinical and histologic findings similar to findings in our HIV-infected patients. The microscopic, ultrastructural, and immunohistochemical characteristics of the bacterium in our patients and the cut-scratch disease bacillus are identical.

We have described the clinical features of four patients with AIDS or AIDS-related complex who developed systemic symptoms and angiomatous lesions of skin and bone. In several of our cases, the lesions were mistaken by clinical and histologic findings for Kaposi sarcoma. A bacterium was identified in these lesions that appears to be identical to the agent causing cat-scratch disease. Antibiotic therapy led to resolution of the lesions and the accompanying systemic symptoms.

Addendum

Drs. Cockerell and Friedman-Kien recently reported (21) finding Warthin-Starry-positive bacteria in their cases of epithelioid angiomyositis. They report the isolation of a gram-negative rod similar to, if not identical to, the cat-scratch disease bacillus from these lesions. Knobler and colleagues also recently described (22) unique vascular skin lesions associated with human immunodeficiency virus. Four specimens were subsequently evaluated with Warthin-Starry staining and three were noted to contain bacteria.

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References