Staphylococcal Pyomyositis in Patients Infected by the Human Immunodeficiency Virus

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PURPOSE: **We describe the manifestations of spontaneous staphylococcal pyomyositis in patients infected by the human immunodeficiency virus (HIV).**

PATIENTS AND METHODS: **We present the courses of five previously unreported patients infected by HIV who presented to our medical centers with spontaneous staphylococcal pyomyositis. Additionally, we review all previously reported cases of this entity in HIV-infected patients and discuss its possible pathogenesis and importance in the context of HIV infection.**

RESULTS: **All patients presented with gradually developing fever and localized pain and swelling without accompanying leukocytosis. Often only scant evidence of local inflammation was found. None of our patients used intravenous drugs, had a history of trauma, had HIV- or zidovudine-related myositis, or had other conditions known to be associated with serious staphylococcal infections. Two patients studied had normal serum levels of all IgG subclasses. Elevated serum IgE, eosinophilic inflammatory infiltrates, or marked peripheral eosinophilia was observed in two patients.**

CONCLUSIONS: **Staphylococcal pyomyositis in HIV-infected patients presents in an indolent fashion, which may delay appropriate diagnosis and treatment. Since staphylococcal pyomyositis is infrequently reported in the United States, the development of 14 such cases (five in this series and nine previously reported) among the first 140,000 cases of acquired immunodeficiency syndrome in this country implies that this patient population is predisposed to this infectious complication. The pathogenesis of this entity is uncertain, but it is notable that HIV-infected patients are commonly colonized by *Staphylococcus aureus* and that neutrophils from HIV-infected patients frequently manifest phagocytic, chemotactic, and oxidative defects, diminished expression of $Fc_{\gamma}RIII$ (CD16) and CR1, and impaired bactericidal activity against *S. aureus*.**

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Pyomyositis, the formation of abscesses in striated muscle, was first described by Scriba [1] in 1885. Most cases occur in indigenous residents of the tropics [2]. The syndrome was first reported in the United States in 1971 [3]. Subsequently, fewer than 50 cases have been reported in the continental U.S. [4].

Pyomyositis was first reported in a British patient infected by the human immunodeficiency virus (HIV) in 1987. This patient had no risk factors traditionally associated with pyomyositis, such as recent trauma, tropical residence, intravenous drug abuse, or diabetes mellitus. Although no organism was grown from the purulent material obtained at surgery, this patient responded to treatment with gentamicin and flucloxacillin [5]. Since that time, 10 additional cases of HIV-related pyomyositis have been reported [4,6–10]. Herein, we describe five additional cases of staphylococcal pyomyositis in HIV-infected patients, review the available literature regarding the characteristics of this entity in HIV-infected patients, and discuss its possible pathogenesis and importance in the context of HIV infection.

CASE REPORTS

Patient 1

A 29-year-old homosexual man with the acquired immunodeficiency syndrome (AIDS) presented with 2 weeks of worsening pain and swelling in the left calf, fever, and mild diarrhea. He denied trauma to the leg, recent travel, or intravenous drug abuse. Six months earlier, a cutaneous lesion of Ka-

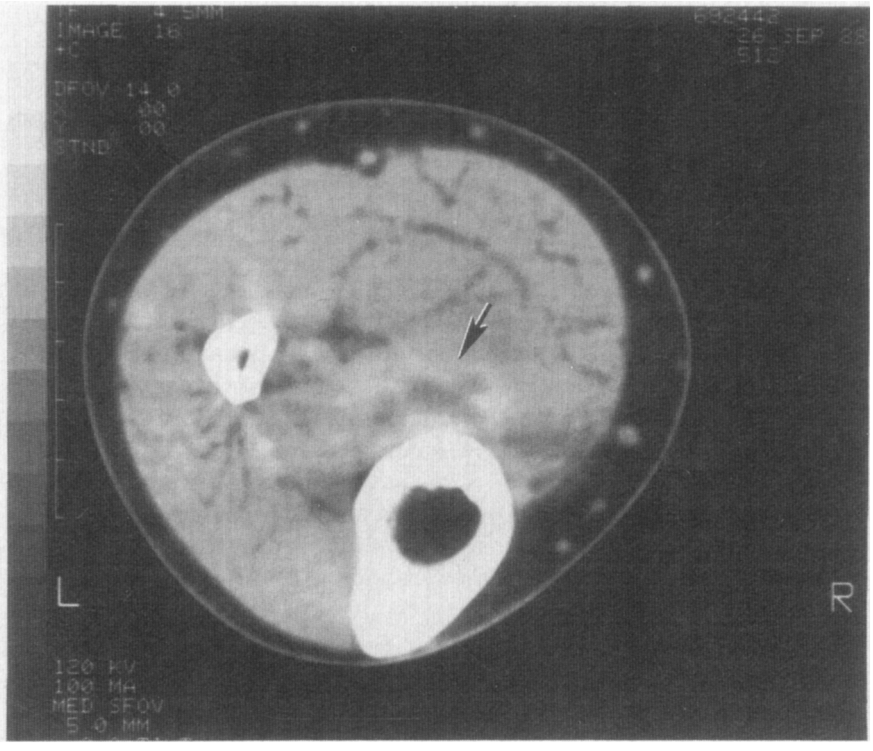

Figure 1. Parenteral contrast-enhanced CT scan of left gastrocnemius of Patient 1 demonstrating area of pyomyositis (**arrow** indicates site of lesion).

posi's sarcoma was noted on his right foot. His course was subsequently complicated by the development of gastrointestinal cryptosporidiosis and by bacteremic *Streptococcus pneumoniae* pneumonia 4 and 2 months prior to his current admission. At the time of presentation, he was receiving zidovudine but no prophylaxis against *Pneumocystis carinii* pneumonia (PCP).

His oral temperature was 38°C, his pulse was 90 beats/minute, and his blood pressure was 130/80 mm Hg. A 2 × 3-cm Kaposi's sarcoma lesion was present on the left anterior tibial surface, and a 5 × 6-cm tender, erythematous, nonfluctuant area was noted on the medial aspect of the left calf. Homans' sign was present but no venous cords were palpable. Normal arterial pulses were present.

The white blood cell count was 1.5×10^9/L with 40% segmented neutrophils. The absolute CD4 count was 0.024×10^9/L and the absolute CD8 count was 0.546×10^9/L. The blood urea nitrogen (BUN), serum creatinine, creatine phosphokinase, lactate dehydrogenase, aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, electrolytes, urinalysis results, and admission chest roentgenogram were normal. Blood cultures showed no growth. Contrast venography of the left lower extremity showed no thrombus. A computed tomographic (CT) scan was done with intravenous contrast and revealed a 3-cm diameter contrast-enhancing lesion with central lucency in the left gastrocnemius (**Figure 1**). This lesion was poorly visible in the nonenhanced study. CT-guided needle aspiration yielded 6 mL of purulent material, which on Gram stain demonstrated numerous polymorphonuclear leukocytes (PMNs) and gram-positive cocci in clusters. *Staphylococcus aureus* grew on culture of this material.

The patient was treated with cefazolin for 4 days followed by vancomycin for 5 additional days. The patient improved rapidly and oral cephalexin was begun on the ninth day of hospitalization. Ten days later, the patient had fully recovered.

Patient 2

A 39-year-old homosexual man with AIDS presented with 2 weeks of severe left calf swelling and pain. There was no history of recent trauma, travel, or intravenous drug use. He had been well until 10 months prior to admission, when he developed PCP. He was subsequently treated with weekly oral sulfadiazine-pyrimethamine for PCP prophylaxis and zidovudine. Although he had been in clinically stable condition for several months prior to this admission, his course had previously been complicated by recurrent episodes of staphylococcal folliculitis with an eosinophilic cellular infiltrate that had responded to oral antibiotics. At the time of presentation, he had not received such therapy for greater than 4 weeks.

His oral temperature was 38.2°C. Homans' sign was positive and the left calf was swollen, erythematous, tender, and slightly warmer than the right. No

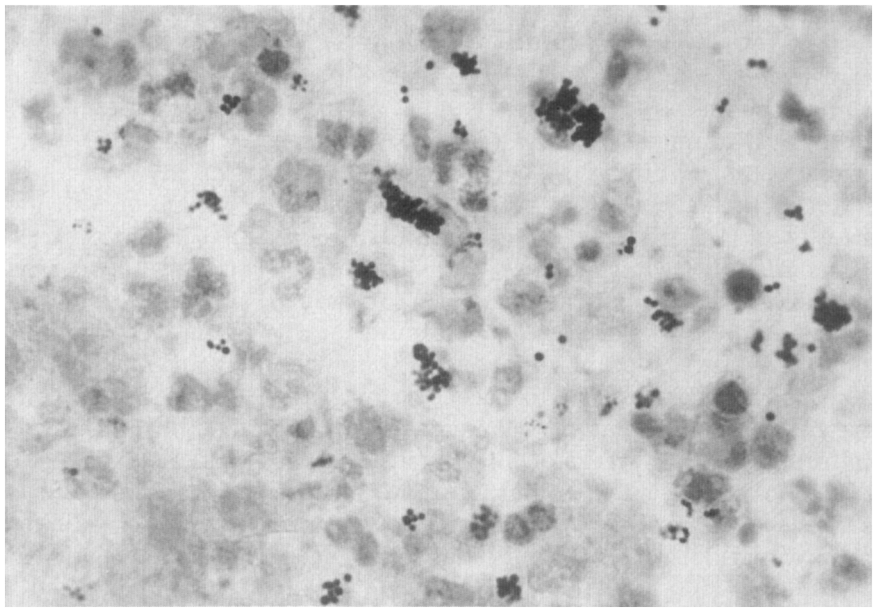

Figure 2. Gram stain of contents of muscle abscess from Patient 5. Note the high ratio of extracellular to intracellular bacteria (original magnification ×1,000, reduced by 30%).

venous cords were palpable and normal arterial pulses were present.

The white blood cell count was 1.5×10^9/L, with 65% segmented neutrophils, 6% band forms, 19% lymphocytes, 9% monocytes, and 1% eosinophils. The BUN, creatinine, electrolytes, urinalysis results, and chest roentgenogram were normal. Other studies demonstrated a serum IgE of 913 IU/mL (normal less than 180 IU/mL) but normal total IgG, IgG subclasses, C3, C4, and CH_{50}. Contrast venography of the left leg revealed no thrombus. CT scan with contrast revealed a 1 × 1.5-cm enhancing lesion with fluid density in the left medial gastrocnemius, which was poorly visible without the administration of parenteral contrast. Aspiration of this collection yielded purulent material containing numerous PMNs and gram-positive cocci in clusters on Gram stain. *S. aureus* was identified on cultures of this material. Blood cultures were repeatedly negative.

The patient was treated with intravenous nafcillin for 1 week followed by oral dicloxacillin for an additional week. He was asymptomatic when seen 2 weeks later. Two months later, he developed acute staphylococcal sinusitis of the frontal and ethmoid sinuses. Subsequently he had separate episodes of staphylococcal folliculitis, periorbital cellulitis, and sinusitis.

Patient 3

A 29-year-old homosexual man with AIDS presented with left chest pain and fever of 2 weeks' duration. He denied chest wall trauma, intravenous drug use, recent travel, diabetes mellitus, or a prior history of staphylococcal skin infections. His past medical history included three prior episodes of biopsy-proven PCP and oral Kaposi's sarcoma lesions. Eight months prior to this presentation, cryptococcal meningitis had been diagnosed. Current medications included phenytoin for seizure prophylaxis, aerosolized pentamidine, and weekly amphotericin B administered via a peripheral intravenous catheter. Zidovudine had been discontinued 2 weeks earlier because of granulocytopenia. An absolute CD4 lymphocyte count 2 months previously was 0.023×10^9/L, with a CD4 to CD8 ratio of less than 0.2.

His temperature was 39°C. A 4.0 × 5.0-cm erythematous mass was noted inferior and lateral to the left areola with indurated margins and an area of central fluctuance. Multiple tender lymph nodes were palpated in the left axilla.

The white blood cell count was 3.6×10^9/L with 47% segmented neutrophils, 5% band forms, 15% lymphocytes, and 27% eosinophils. Levels of serum electrolytes, BUN, and creatinine were normal. Aspiration of the mass yielded 3.5 mL of purulent material, and Gram stain showed gram-positive cocci in clusters with numerous PMNs; cultures grew *S. aureus*. Incision and drainage demonstrated frank pus and necrotic subcutaneous tissue overlying the left pectoral fascia; the pectoralis major muscle showed focal areas of hemorrhage with multiple abscess cavities. The patient received intravenous oxacillin perioperatively followed by oral dicloxacillin for a total of 21 days. The wound was packed and healed by secondary intention.

Patient 4

A 33-year-old homosexual man with AIDS who denied trauma or intravenous drug use presented with several days of fever, night sweats, and severe

pain in the left hip. HIV infection and disseminated histoplasmosis had been diagnosed 17 months earlier. Current medications included only ketoconazole and zidovudine.

Physical examination revealed decreased range of motion of the left hip due to pain without swelling or erythema. His white blood cell count was 8.3 $\times 10^9$/L and the erythrocyte sedimentation rate was 105 mm/hour. Roentgenography revealed soft tissue swelling of the gluteal region, and sonography revealed a fluid-filled mass that was subsequently aspirated, yielding purulent material. Microscopy demonstrated gram-positive cocci, and cultures grew *S. aureus*. The patient was treated with surgical drainage and 2 weeks of intravenous cephalothin followed by 8 weeks of oral cephalexin. He had a rapid resolution of symptoms and no recurrence of pyomyositis during 6 weeks of follow-up.

Patient 5

A 40-year-old homosexual man with AIDS who denied trauma or intravenous drug use presented with 3 weeks of worsening pain in the left calf with swelling, erythema, and low-grade fever. One year earlier, he had been found to have Kaposi's sarcoma, hairy leukoplakia, and a CD4 lymphocyte count less than 0.1 $\times 10^9$/L. He had been treated with radiation therapy to the face and trunk for progressive Kaposi's sarcoma. The patient had also been given systemic vincristine and vinblastine to treat suspected pulmonary Kaposi's sarcoma lesions. Current medications included zidovudine and aerosolized pentamidine.

The patient was initially evaluated with sonography 2 weeks after the onset of his symptoms. This study revealed a 5 × 6-cm fluid density within the body of the muscle believed to be consistent with hematoma. At this time, his white blood cell count was 9.1 $\times 10^9$/L. Serum concentrations of all IgG subclasses were normal. The patient was treated symptomatically. Three days later, the patient developed fever to 40°C and worsening pain. Needle aspiration of the lesion revealed frank pus and numerous extracellular and intracellular gram-positive cocci on microscopic examination (**Figure 2**). Cultures of the aspirate were positive for *S. aureus* but blood cultures were negative. The patient had an excellent response to surgical drainage and treatment with intravenous nafcillin for 6 days followed by oral dicloxacillin for 4 weeks. The patient was then found to have cryptoporidiosis and died 1 month later of complications of this illness.

COMMENTS

Pyomyositis is very uncommon in temperate climates. An extensive review in 1988 found only 47 cases from the continental U.S. [4]. The development of 16 cases of pyomyositis in HIV-infected patients ([4–10] and this series), all but two of which [5,6] occurred among the first 140,000 cases of AIDS reported in the U.S. [11], indicates that patients with AIDS are substantially predisposed to develop this infection.

None of our patients used intravenous drugs, had a history of trauma, had HIV- or zidovudine-related myositis [12,13], or had other conditions known to be associated with serious staphylococcal infections. Although human T-cell lymphotropic virus type II infection has been recently associated with a predilection to serious staphylococcal soft tissue infections in intravenous drug users [14], immunoblot analyses of serum from Patients 2 and 5 showed no evidence of such infection. Finally, we doubt that the weekly administration of amphotericin B via a peripheral catheter was the primary cause of staphylococcal pyomyositis in Patient 3. Although therapeutically achievable concentrations of amphotericin B impair neutrophil chemotaxis, phagocytosis, and bactericidal activity against *S. aureus in vitro* [15,16], we know of no clinical data that directly link the use of this antifungal agent to the development of serious infections.

The clinical manifestations of pyomyositis in our five patients were notable for the subacute development of pain, swelling, and fever. Erythema was an unreliable finding in these cases, and leukocytosis or laboratory evidence of muscle necrosis was not found. *S. aureus* was cultured from abscesses in all of our cases. Blood cultures were uniformly negative when done. All cases resolved with antimicrobial therapy directed against *S. aureus* combined with open (three cases) or closed (two cases) drainage.

Ten of the 11 previously reported cases of pyomyositis in HIV-infected patients involved adult males and the other occurred in a 3½-month-old infant [4–10]. Two of these cases did not meet criteria for the diagnosis of AIDS. One patient had no HIV-related symptoms and the other was an intravenous drug user with thrush, recent weight loss, and a CD4 to CD8 ratio of 0.03 [5,6]. Aside from two patients who had a history of intravenous drug use [6,9], none of the previously reported HIV-infected patients had any predisposition for developing pyomyositis. All patients had fever, swelling, and pain, and an elevated erythrocyte sedimentation rate where reported. Other physical and laboratory findings were not helpful. Cultures of the lesions grew *S. aureus* in eight cases; no pathogen was identified in the other two patients. Positive blood cultures have only been reported once [9]. All these patients responded to therapy with antimicrobial agents, although open drainage was also utilized in nine cases.

Other diagnostic considerations in HIV-infected patients with focal calf pain and swelling include HIV-related "hyperalgesic pseudothrombophlebitis" [17]. Of the five reported patients with this syndrome, four were febrile, three had elevated erythrocyte sedimentation rates, and four were treated with antibiotics. Although one had a normal CT scan, contrast enhancement was not utilized. On the basis of these data, we speculate that some of the reported cases of "hyperalgesic pseudothrombophlebitis" may in fact have been cases of HIV-associated pyomyositis.

Defects of neutrophil function may contribute to the development of staphylococcal pyomyositis in some HIV-infected patients. PMNs from HIV-infected patients frequently manifest decreased chemotaxis [18–25], expression of $Fc_{\gamma}RIII$ (CD16) [26] and CR1 [27], oxidative metabolism [19,21,24,28,29], bactericidal activity against *S. aureus* [23,25,28,30], and ability to bind or ingest *Candida albicans* [22,23] or *S. aureus* [30]. These PMN defects do not clearly correlate with the stage of HIV infection [23,24,27] and apparently reflect intrinsic cellular defects as well as serum-mediated effects [19–21,24,27,28,31].

The means by which HIV infection may affect PMN function are unknown. HIV does not infect mature PMNs [32], but the envelope glycoprotein GP-160 inhibits PMN chemotaxis [33], and alterations in the concentrations of circulating cytokines associated with HIV infection may disrupt PMN function [34,35]. Interestingly, marked elevations of IgE not infrequently accompany the advanced stage of HIV disease [36–40]. Increased serum IgE levels, as observed in persons with atopic dermatitis and hyperimmunoglobulinemia E (Job's syndrome), are associated with decreased PMN chemotaxis [41,42], decreased PMN C3b receptors [43], "cold" staphylococcal abscesses [41,42,44], and eosinophilic inflammatory infiltrates [42]. Similarly, the serum IgE level in Patient 2 was five times the upper limit of normal; an eosinophilic infiltrate developed in response to one episode of staphylococcal infection in this patient, and a marked peripheral eosinophilia accompanied the episode of pyomyositis in Patient 3. Furthermore, our patients often manifested scant evidence of local inflammation. Although the pathophysiology of hyperimmunoglobulinemia E is uncertain, it appears that the massive increases of serum IgE are not the primary cause of PMN dysfunction but rather reflect underlying immune dysregulation [40,45–49].

In summary, staphylococcal pyomyositis appears to be a complication of HIV infection. Previous reports indicate that HIV-infected patients are frequently colonized by *S. aureus* [50] and are prone to develop recurrent episodes of *S. aureus* folliculitis [20], pneumonia [51], and metastatic infections consequent to *S. aureus* bacteremia [52]. We believe that HIV-infected patients with muscular swelling, localized pain, and fever should be promptly evaluated with ultrasound or by contrast-enhanced CT scanning [53,54]. If a fluid collection is visualized, diagnostic needle aspiration should be performed. Empiric antistaphylococcal therapy combined with closed or open surgical drainage is warranted if purulent material is found. In patients with recurrent episodes of deep-seated staphylococcal infections, it may be appropriate to attempt to eradicate *S. aureus* colonization.

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