Cutaneous Leishmaniasis following Local Trauma:  
A Clinical Pearl

Cutaneous leishmaniasis is acquired from the bite of an infected sand fly and can result in chronic skin lesions that develop within weeks to months after a bite. Local trauma has been implicated as a precipitating event in the development of skin lesions in patients who have been infected with Leishmania species. Here we report a case series and review the literature on patients who developed cutaneous leishmaniasis after local trauma, which may familiarize clinicians with this presentation.

New World cutaneous leishmaniasis is acquired from the bite of infected female sand flies. The skin lesions typically evolve over weeks to months from papules to nodules to ulcers with raised, indurated borders [1]. The time from the bite to the development of a lesion varies; incubation periods range from days to months [2]. The ulcerative skin lesion seemingly precipitated by local trauma is a clinical pearl associated with this disease. In 1974, Walton and Valverde reported a series of 4 American soldiers in Panama who had a defined history of exposure to Leishmania species in an area of endemcity, followed by an extended period living in a nonendemic location. After sustaining relatively minor trauma (a puncture wound of the hand, an eyelid struck by a small piece of gravel, a pimple that was manipulated, and a repeatedly abraded elbow) each patient developed a leishmanial lesion at the site of trauma [3]. In a different publication, Walton presented a dramatic photograph of a leishmanial lesion that developed along the margins of the length of a laceration [4].

In addition to these reports, cutaneous leishmaniasis that developed at the site of an injury has been reported after a bump on the forehead sustained after striking a low hanging beam [5], a coral cut on the palm [6], a creosote burn on the nose [7], and after a cat scratch [8]. This phenomenon does not appear limited to New World disease, however, as cutaneous leishmaniasis has been reported following submucous nasal resection (Leishmania major) [9], in the herpes zoster lesions of an HIV-infected patient in Spain (species not reported) [10], and after a skin snip biopsy of a patient with visceral disease (Leishmania infantum) [11]. After evaluating a patient who developed cutaneous leishmaniasis at the site of a recently placed tattoo, we reviewed our experience with patients who developed cutaneous leishmaniasis at sites of local trauma.

We reviewed medical records for patients diagnosed with cutaneous leishmaniasis (defined as evaluation of a skin lesion biopsy demonstrating amastigotes on histopathology or growth of promastigotes in culture of the biopsy) who were evaluated at the Walter Reed Army Medical Center (Washington, DC) from 1994 through 1999, in order to identify physician notes stating that skin lesions occurred after local trauma. Medical histories were unstructured and this association was not specifically solicited. Seven patients were identified whose cutaneous lesions were seemingly precipitated by local trauma. Six of 7 patients acquired their disease in Panama while participating in training at the military’s jungle training school. In 6 cases, leishmanial culture of the cutaneous lesion allowed characterization by analysis of isoenzyme patterns in electrophoresis with cellulose acetate [12, 13]. All patients received treatment with iv sodium stibogluconate (Pentostam; Glaxo-Wellcome, London) with clinical cure of their lesions.

Case 1 was in a 21-year-old man stationed in Panama who developed an ulcerative lesion in the right temporal area and was given an oral antibiotic without effect. One month later, he was reevaluated because of the same lesion and was given a different antibiotic, again without effect. Several weeks later, the patient received a circumferential tattoo over the left biceps. Within 5 days of placement of the tattoo, the patient developed several ulcerative lesions at the site of the tattoo. He also developed a lesion on his right thigh, which was biopsied and cultures yielded Leishmania (Viannia) panamensis.
Case 2 was in a 26-year-old man who was stationed in Panama for ~1 month during which time he had no complaints. Two months after returning to the United States, the patient cut himself while shaving. An ulcerative skin lesion rapidly developed at the site over the next few days. Culture of a biopsy specimen of the lesion yielded Leishmania (V.) braziliensis.

Case 3 was in an 18-year-old man who was stationed in Panama for 4 months. He was stung by a bee on the forehead ~1 month after returning to the United States. A short while later (the precise time period was not recorded in the chart), a nodule developed that progressed to an ulcerative skin lesion. Culture of a biopsy specimen of the lesion yielded L. (V.) panamensis.

Case 4 was in a 19-year-old man who was stationed in Panama for 4 months. He developed an ulcerative skin lesion on the right wrist. He subsequently sustained minor puncture wounds to the right calf by a rifle and the left thigh by a branch, and ulcerative lesions developed shortly after (the precise period of time was not recorded in the chart) at both sites. Histopathologic evaluation of a wrist lesion biopsy was positive for amastigotes, but culture of the specimen yielded no growth.

Case 5 was in a 20-year-old man who was stationed in Panama for ~1 month and noted a small papule on his left cheek toward the end of his tour. Three weeks after returning to the United States the patient abraded this papule while playing rugby. An ulcerative lesion subsequently developed at the site, and culture of a biopsy specimen of the lesion yielded L. (V.) panamensis.

Case 6 was in a 20-year-old man who was stationed in Panama for ~1 month without complaint. After returning to the United States, the patient accidentally cut a small nodule on his leg while shaving. An ulcerative lesion developed at the site, and culture of a biopsy specimen yielded L. (V.) panamensis.

Case 7 was in a 23-year-old man who, when he returned from Panama for 4 months, he developed an ulcerative skin lesion on the right wrist. He subsequently sustained minor puncture wounds to the right calf by a ri®e and the left thigh by a branch, and ulcerative lesions developed shortly after (the precise period of time was not recorded in the chart) at both sites. Histopathologic evaluation of a wrist lesion biopsy was positive for amastigotes, but culture of the specimen yielded no growth.

In summary, local trauma appears to be associated with the development of skin lesions in patients infected with Leishmania species. This observation may help clinicians consider the diagnosis in patients who offer a compatible history. This finding may also have clinical implications when advising patients who are planning elective surgery, and we counsel our patients to postpone surgery until after they’ve received adequate treatment for leishmaniasis.

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Helicobacter cinaedi Septic Arthritis and Bacteremia in an Immunocompetent Patient

We report on the first case of documented Helicobacter cinaedi septic arthritis in an immunocompetent heterosexual young man. The patient presented no identified risk factor except for contact with animals that have been in-
criminated as a possible source of infection, particularly for these patients. Despite prolonged bacteremia, the re-
sponse to long-term therapy with ciprofloxacin and rifampin was excellent.

Helicobacter cinaedi (previously called Campylobacter-like organism) was first isolated from rectal swabs of homosexual men with proctocolitis [1]. H. cinaedi is also responsible for bacteremia complicated with cellulitis or monoarticular ar-
thritis. Forty-seven cases of H. cinaedi bacteremia have been reported, most of which occurred in HIV-infected homosexual men or in patients with underlying immunosuppressive factors (e.g., alcoholism, cancer, immunosuppressive therapy, preg-
nancy, or neonate) [2–5]. Only 3 cases of H. cinaedi infection have been observed in heterosexual immunocompetent men [3]. We report a case of H. cinaedi bacteremia and septic arthritis in an otherwise healthy immunocompetent man and discuss the clinical and laboratory features pertinent to its recognition.

A 20-year-old man was hospitalized in August 1998 with a 24-h history of fever (temperature, 38.1°C) and an acute in-
mflammation of the right knee with synovial effusion. The re-
mainder of the physical examination was normal except for a scab on the anterior surface of the opposite knee. Radiography of the right knee was normal. Laboratory findings showed a
WBC count of 13,600 cells/µL (77% neutrophils) and a high level of C-reactive protein (98 mg/L). There was no humoral immunity defect, and HIV serology was negative. The micro-
scopic examination of the right knee joint fluid showed 25,000 nucleated cells/µL (85% neutrophils), but no bacteria were seen. Bacterial culture was performed on the joint liquid and blood samples on solid media and by inoculation in Bact-Alert bottles (Organon Teknika, Durham, the Netherlands). After 36–72 h of incubation, aerobic bottles with blood and joint fluid flagged positive, whereas cultures on solid media remained negative, even after a 5-day incubation. The microscope examination revealed a motile, 10-µ–spiralated, faintly gram-negative bac-
terium. It mimicked a spirochete, but Lyme serology and Borrelia burgdorferi PCR were negative.

Subcultures from Bact-Alert bottles yielded growth on Camp-
ylobacter selective media after a 4-day incubation under microaerobic conditions. However, further subcultures showed no growth. Biochemical test results revealed the organism to be oxidase-positive, catalase-positive, and nitrate reductase-positive, but urease and hippurate hydrolysis were negative; this suggested that the bacterium was H. cinaedi. Complete iden-
tification and susceptibility testing using a disk diffusion tech-
nique was performed by the French National Reference Center of Campylobacter and Helicobacter. The isolate was susceptible to nalidixic acid, rifampin, and tetracycline but resistant to erythromycin and cephalotin. The 16S rRNA gene nucleotide sequence, determined as described elsewhere [6], confirmed the identification.

Antimicrobial therapy against spirochete (iv penicillin G, 12 million U/d) was first initiated in combination with analgesics, joint immobilization, and drainage. When an H. cinaedi infec-
tion was suspected, ciprofloxacin (1g/d) and rifampin (1.2 g/d) were given for 12 weeks (because of the severity of infection and the delay in making the diagnosis). After 2 weeks of treat-
ment, the joint fluid became sterile and the synovitis of the knee disappeared without any joint sequelae.