Bacillary angiomatosis

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CASE PRESENTATION

DR J. SHAUN MURPHY: A 30-year-old woman was found to have human immunodeficiency virus (HIV) at age 22 (1989). She also had a history of moderate asthma. She had been in her usual health until 2 months before hospitalization when nonbloody diarrhea appeared. A month later, she noted a small, yellow mass arising from her umbilicus (Figure 1). Seven days before the patient’s admission to the hospital, the umbilical mass was noted to drain small amounts of clear fluid. In addition, she noted the onset of exertional dyspnea, diffuse abdominal pain, increasing abdominal girth, fever, chills, urinary frequency, dull lower back pain, and general fatigue.

The HIV was secondary to intravenous drug use. She stated she had not used heroin or cocaine intravenously since 1995. Eight months before admission, her CD4 count was 12 cells/mm³. Two years before admission, she had candidiasis and herpes zoster. The asthma was controlled by metered-dose inhalers. Both fallopian tubes had been ligated when she was 22. Prior to that, she had had 3 babies, 1 of whom was known to be HIV positive. She had smoked >20 cigarettes daily for many years. She drank 2 cases of beer each week. She was unemployed but occasionally volunteered at a local animal shelter.

On admission, the patient was weak, fatigued, and febrile. She had the umbilical mass and no rash. She denied blurred or double vision, and she had no photophobia. She had no vertigo or hearing difficulties. She had no sinus pain or nasal drainage. She had many false teeth. Her neck and breasts appeared normal. She had a cough with a little nonbloody sputum. She denied chest pain and nocturnal dyspnea. She had nonbloody diarrhea and urinary frequency but no dysuria, hematuria, or urgency. She did not have arthralgia, arthritis, or claudication. She denied headaches, a history of seizures, blackouts, numbness, tingling, and paralysis. She had no known drug allergies. She used albuterol metered-dose inhalers and was taking zidovudine, 300 mg twice daily; lamivudine, 150 mg twice daily; and indinavir, 800 mg 3 times daily. Her temperature was 37.3°C (98.9°F); blood pressure, 80/50 mm Hg; heart rate, 100 beats per minute; and respiratory rate, 22.

She appeared ill and in mild respiratory distress. She was normocephalic; extraocular eye movements were intact; and her pupils were bilaterally equal, round, and reactive to light and accommodation. Her fundi and tympanic membranes were normal. Her mucous membranes were dry. She had very poor dentition. Her neck was supple, and there was no jugulovenous distension or lymphadenopathy. The thyroid gland was of normal size. There was no carotid bruit. The breasts
were symmetrical without masses or discharge. No lymph nodes were palpable in the axilla or elsewhere. Her lungs were clear to auscultation. Precordial examination disclosed no abnormalities. Her abdomen was protuberant, with a 5-cm draining mass in the umbilicus. The bowel sounds were normal, and no bruits were heard. Her liver was palpated 10 cm below the costal margin, and the caudal margin of the spleen also was palpated. Rectal examination disclosed no masses. Stool specimen was guaiac negative. Neurological examination showed that cranial nerves 2–12 were intact. Sensation and cerebellar function were intact. Strength was 5 out of 5 bilaterally, and she had 2+/4+ deep tendon reflexes throughout.

The white blood cell count was 4400/?L; hemoglobin, 7.2 g/dL; hematocrit, 21.1%; mean corpuscular volume, 70 fL; platelet count, 116,000/?L. White blood cell differential included 72% segmented neutrophils, 22% bands, 4% lymphocytes, and 2% monocytes. Reticulocyte count was 0.9; total iron binding capacity, 178 ?g/dL; iron saturation, 12%; serum iron, 21 ?g/dL; serum ferritin, 1734 ng/mL; prothrombin time, 11.8 seconds; partial thromboplastin time, 35.1 seconds; bleeding time, 5.5 minutes. Other laboratory results were glucose, 89 mg/dL; sodium, 123 mEq/L; potassium, 3.7 mEq/L; chloride, 91 mEq/L; CO2, 24 mg/dL; blood urea nitrogen, 10 mg/dL; creatinine, 1.1 mg/dL; total cholesterol, 109 mg/dL; triglycerides, 186 mg/dL; uric acid, 9.4 mg/dL; phosphorus, 3.7 mg/dL; calcium, 7.5 mg/dL; total protein, 5.0 g/dL; albumin, 2.2 g/dL; total bilirubin, 4.6 mg/dL; direct bilirubin, 3.1 mg/dL; alkaline phosphatase, 327 U/L; y-glutamyltransferase, 658 U/L; aspartate aminotransferase, 103 U/L; alanine aminotransferase, 46 U/L; lactate dehydrogenase, 243 U/L; creatine phosphokinase, <20 U/L; plasma osmolality, 256 mOsm/kg H2O. Urinalysis showed specific gravity <1.005; pH, 6.5; positive for bilirubin; trace leukocyte esterase; few epithelial cells; no white blood cells; no red blood cells; and light bacteria. Urine chloride was <10 mEq/L and urine sodium, <5 mEq/L. Her CD4 count was 1 cell/mm3. Absolute lymphocytes were 122. Human immunodeficiency virus/polymerase chain reaction titer was 18,769 copies/mL. Hepatitis serology was positive for hepatitis C and negative for hepatitis B. Rapid plasma reagin was nonreactive. Carcinoembryonic antigen was 2.0. Alpha-fetoprotein was 6.6; CA-125 was 95.5. The cutaneous necrotizing vasculitis antigen was negative. Blood cultures were negative for bacterial, mycobacterial, fungal, and viral organisms.

An abdominal ultrasound revealed hepatomegaly with parenchymal heterogeneity and scattered, approximately 1 cm–sized, hypoechoic foci throughout the liver. There was splenomegaly, with a maximum size of 16 cm, and a small amount of ascites. The gallbladder had stones, the wall was thickened, and a small porta hepatis lymph node was seen, as well as the umbilical mass. An abdominal computed tomography showed a profusely abnormal liver, with scattered, 1 cm–sized areas of increased density, splenomegaly, umbilical mass, ascites, gallstones, and a small right pleural effusion.

**DISCUSSION OF RADIOLOGICAL FINDINGS**

DR. ANTHONY C. TOPPINS: A contrast-enhanced computed tomography scan of the abdomen demonstrated diffuse heterogeneity of the hepatic parenchyma (*Figure 2*). There were multiple small foci of increased attenuation throughout both hepatic lobes, without a dominant mass. Although a noncontrast study was not available, the appearance of the hepatic parenchyma suggested a diffuse, hypervascular process. There also was evidence of nonspecific retroperitoneal
adenopathy, splenomegaly, and a small amount of ascites. Finally, there was an exophytic, somewhat hypervascular mass arising from the umbilicus (Figure 3).

CASE DISCUSSION

DR. ANDREW D. CHUNG: In summary, we have a patient with acquired immunodeficiency syndrome (AIDS) who had fever, abdominal pain, hepatosplenomegaly, multifocal hepatic lesions, and a subcutaneous umbilical mass.

A literature search on AIDS, hepatosplenomegaly, and multifocal hepatic lesions revealed a list of articles. Basically, the differential diagnoses included infectious and neoplastic etiologies. Some of the unlikely possibilities included were candidiasis (1), non-Hodgkin's lymphoma (1–3), hepatocellular cancer (4), ovarian cancer, hepatic Schistosoma mansoni (5), hemangiomas (1), Strongyloides stercoralis (6), and Cowden's disease (7). Most of these diagnoses can be eliminated because of discrepancies in the clinical presentation, laboratory results, or radiographic findings.

I would like to focus my discussion on the major differential diagnoses of disseminated cytomegalovirus (CMV) (1, 8, 9), histoplasmosis (1, 4), Pneumocystis carinii pneumonia (PCP) (1, 4), Mycobacterium avium complex (MAC)/Mycobacterium tuberculosis (MTb) (1, 4, 9), bacillary angiomatosis (BA) (1, 4, 10–13), Kaposi's sarcoma (1, 4, 9, 14), and primary effusion lymphomas.

The first diagnosis I would like to consider is CMV. We need to consider this infection in our differential because CMV infection presents when the CD4 count is <100 cells/mm³, and it is the most common life-threatening opportunistic infection in an AIDS patient (1). A person with CMV usually presents with fever and mononucleosis-like symptoms, and this infection can involve any organ, including the liver and spleen. Usually there is leukopenia, atypical lymphocytes, and a positive CMV antigen. Radiographically, ultrasound shows multiple discrete hyperechoic areas secondary to CMV. These areas are caused by inflammatory and fatty infiltration of the liver (8). Our patient had a negative CMV antigen, and the ultrasound showed multiple, small hypoechoic lesions throughout the liver. It is unlikely that CMV infection can explain most of our patient's current illness.

The next diagnosis to consider is histoplasmosis. The fungus Histoplasma capsulatum is endemic to Texas. Histoplasmosis is the most common systemic fungal infection in the USA and can present in patients who have a CD4 count <100 cells/mm³. Patients who have AIDS and disseminated histoplasmosis are very sick and commonly present with fever, diarrhea, abdominal pain, ascites, hepatosplenomegaly, leukopenia, anemia, elevated alkaline phosphatase, elevated alanine aminotransferase, hyponatremia, and cutaneous lesions. The cutaneous manifestations occur in 10% of AIDS patients with disseminated histoplasmosis and are typically erythematous, maculopapular, petechial, and multiple. The computed tomography scan of the abdomen shows hepatosplenomegaly, enlargement of the lymph nodes (>40%), and either diffusely abnormal liver and spleen with low attenuation or multifocal, hypodense lesions in the liver (16). The adrenal glands are enlarged in >80% of patients.

Our patient had many clinical and laboratory findings similar to those associated with disseminated histoplasmosis. Our patient, however, did not have adenopathy, and the skin lesion at the umbilicus
was not consistent with the typical histoplasmosis lesion. The serum potassium, bicarbonate, and alanine aminotransferase were normal. Radiographically, the computed tomography scan of our AIDS patient revealed hyperdense lesions and not hypodense lesions in the liver. Although disseminated histoplasmosis must be considered, because of these discrepancies it is not responsible for all of our patient's symptoms.

The next diagnosis to consider is **disseminated PCP infection** that affects about 85% of patients with AIDS at some point in their illness (17). The high frequency of infection in AIDS patients with a CD4 count of <200 cells/mm³ and the clinical presentation make disseminated PCP infection high on the differential diagnosis. Our patient presented in mild respiratory distress and with tachycardia. There was no information about her oxygenation or results of her chest radiograph, although a normal chest radiograph does not rule out PCP infection (18). In addition, extrapulmonary PCP infection usually occurs in AIDS patients who are taking aerosolized pentamidine for PCP prophylaxis or in patients who are not taking any prophylaxis at all. Patients with HIV infection who develop extrapulmonary pneumocystosis frequently do not have concurrent PCP (17). Patients with extrapulmonary PCP infection can present with fever, abdominal pain, ascites (19), hepatosplenicomegaly, and diarrhea. Extrapulmonary PCP also can manifest as a single lesion on the skin, as a fleshy fungating mass, or as a vegetative lesion with serosanguineous drainage (17). Laboratory results can reveal normal lactate dehydrogenase (21), slightly high lactate dehydrogenase, elevated γ-glutamyltransferase, elevated alkaline phosphatase, and anemia (17). The chest radiograph also can demonstrate pleural effusions, although this is rare (18). Our patient had a clinical picture consistent with disseminated PCP infection. A major discrepancy against this diagnosis is that disseminated PCP in the liver usually presents as numerous hypodense lesions and punctate calcific deposits in the spleen, liver, kidney, and adrenal gland (19). There are no reported cases of disseminated PCP appearing as a hyperdense lesion on the contrast-enhanced computed tomography scan (1).

Another likely diagnosis that can explain our patient's presentation is **disseminated MAC and MTb infection**. Clinically and radiologically, MAC and MTb have subtle differences, and, because of the clinical and radiologic overlap, cultures are needed to differentiate them (20). Disseminated MAC and MTb can become a problem when the CD4 count is <200 cells/mm³. Clinically, AIDS patients will present with fever, abdominal pain, diarrhea, hepatosplenomegaly, ascites (21), profound anemia, elevated alkaline phosphatase, and cutaneous manifestations that can be a single lesion (21). Disseminated MAC and MTb can result in a single, nontender, enlarged lymph node in the inguinal and femoral areas (21). There are no reported cases of disseminated MAC and MTb causing an enlarged lymph node in the umbilicus. The dermal lymphatic vessels of the umbilicus, however, communicate with the subserosal lymphatic network through the inguinal and para-aortic nodes (22). Therefore, theoretically, infection can spread via these channels during retrograde lymph flow (22). The lesion can be fleshy and fungated and can ulcerate and drain serosanguineous fluid, just like our patient's lesion (21). In our patient, every clinical and laboratory finding is consistent with disseminated MAC or MTb. Even the CA-125 has been shown to be elevated in a disseminated MTb infection (23). Our patient is a strong candidate for having a disseminated MAC and MTb infection.

On the other hand, there are some findings that are puzzling and sway against this infection. Although abdominal adenopathy can be sparse in disseminated MAC or MTb, there is always
significant abdominal adenopathy in MAC or MTb peritonitis (21). Our patient had only 1 enlarged peri–porta hepatitis node, which is weak evidence for a MAC or MTb intra-abdominal infection. Another important discrepancy is that disseminated MTb and MAC appear as low-attenuation densities and not hyperdensities (1). Our patient had hyperdensities in the liver. In early MAC or MTb infection, adenopathy may be absent, and time is needed for calcific deposits to occur. Our patient may have an early disseminated MAC or MTb infection and should be treated as such until proved otherwise.

The last infectious etiology on my differential is BA. This infection can become a problem in AIDS patients who have CD4 counts <200 cells/mm³. The clinical manifestations include fever, abdominal pain, diarrhea, hepatosplenomegaly, ascites, pleural effusions, and weight loss (24). The skin lesions of BA can be cutaneous or subcutaneous. Cutaneous lesions look like Kaposi's lesions, are often papular and red, and have a vascular appearance (12). Subcutaneous lesions are flesh-colored nodules that may erode through the surface and become friable and superinfected (12). Manifestation as a single, deep, soft-tissue mass with normal-appearing underlying skin also has been reported (10, 12, 24). Laboratory tests reveal nonspecific findings such as elevated alkaline phosphatase, elevated \( \gamma \)-glutamyltransferase, anemia, leukocytosis, and elevated transaminases. Frequently, the blood cultures are negative (10, 24, 25). Radiographic evidence of BA includes hypoechoic lesions <1 cm in the liver or spleen by ultrasound and hyperdense lesions on contrast-enhanced computed tomography with or without lymphadenopathy (11, 25). There is a case report of a patient with BA, and the only adenopathy present was in the porta hepatis area, just as in our patient (25). Everything in our patient's clinical presentation, laboratory results, and radiographic studies suggests BA, even the history of her working in the animal shelter (10).

I would like to shift gears, because AIDS patients commonly present with more than one diagnosis. I found a case report of a patient who presented very similarly to our patient. He was a 32-year-old man with AIDS who had 6 weeks of increasing abdominal girth, diarrhea, weight loss, cough, fever, ascites, hepatosplenomegaly, and adenopathy. His chest radiograph showed a right pleural effusion, and an abnormal abdominal computed tomography scan revealed ascites, hepatosplenomegaly, and multiple hypodense lesions in the liver and spleen. This other patient ended up having active PCP, \textit{Hemophilus} influenza, CMV, Kaposi's sarcoma, and BA. He was treated immediately with multiple drugs (13).

Because AIDS patients commonly present with more than one diagnosis, the neoplastic etiologies that also can mimic our patient's presentation need to be considered. The 2 most likely neoplastic processes that could help explain our patient's presentation are disseminated Kaposi's sarcoma and primary effusion lymphoma. Patients with AIDS have approximately a 40% chance that cancer will develop in their lifetimes, especially \textit{Kaposi's sarcoma} (26). Kaposi's sarcoma can become a problem when the CD4 is <200 cells/mm³. Kaposi's sarcoma can affect the liver and gastrointestinal tract, producing diarrhea and weight loss. In some case studies, Kaposi's sarcoma was found in the liver of 20% of AIDS patients on autopsy; the neoplasm can occur diffusely in the liver, and the patient can remain asymptomatic (13). The cutaneous lesions are nodular, pigmented, and violaceous. Kaposi skin lesions also can be subcutaneous lesions that are nonpigmented, exactly like our patient's umbilical mass (27).
Sarcoma, but, more commonly, carcinoma (mostly of the gastrointestinal and g astrourinary tract) can metastasize to the umbilicus (28). The most frequent sites of the primary carcinoma are the stomach, liver, colon, ovaries, and endometrium (28, 29). A metastatic nodule in the umbilicus is sometimes referred to as a Sister Mary Joseph's nodule. Sister Mary Joseph, who, in 1912, was a nurse superintendent at the Mayo Clinic, noted the prognostic significance of these lesions. In 1960, Hamilton Bailey suggested that metastatic tumors of the umbilicus should be given the name Sister Mary Joseph's nodule (30). Our patient had pleural effusion, ascites, and hepatic lesions consistent with metastatic cancer, a possible Sister Mary Joseph's nodule, and an elevated CA-125. Ovarian cancer would be high on the differential, if the pelvic computed tomography scan revealed abnormal ovaries.

The last malignancy in my differential is primary effusion lymphomas, well described in the journal Blood in 1996 (31). These lymphomas are identified with Kaposi's sarcoma–associated herpesvirus (26) and are body-cavity lymphomas that usually grow exclusively in the pleural, pericardial, or peritoneal cavities as lymphomatous effusions in the absence of identifiable tumor mass (26, 31). Our patient had a pleural effusion and ascites that could be secondary to primary effusion lymphoma with liver involvement, Kaposi's sarcoma, or an infectious agent.

In conclusion, my differential diagnosis included the most common infectious and neoplastic processes likely to be responsible for our patient's presentation. Based on the clinical presentation, laboratory data, and radiographic findings, the most likely primary diagnosis is bacillary angiomatosis.

**DISCUSSION OF PATHOLOGICAL FINDINGS**

DR. CINDA PARKER: Two similar punch biopsies from the umbilical lesion showed surface ulceration and necrotic debris with an underlying band of fibrosis. Deep to that was a vascular proliferation that consisted of ill-defined lobules of capillaries. These capillary spaces were dilated and rounded and had very protuberant and cuboidal endothelial cells. Also within the stroma were epithelioid cells, some of which had intracytoplasmic vacuoles that also indicated their vascular epithelial nature (Figure 4). Throughout the stroma there was a prominent inflammatory infiltrate that consisted mostly of polymorphonuclear neutrophils. These neutrophils were centered predominantly around granular purple clusters (Figure 5).

The major differential includes the very common pyogenic granuloma, also called lobular capillary hemangioma. Pyogenic granulomas, however, tend to have more jagged, ectatic lumina instead of the rounded ones seen in this case. Also, some vessels in pyogenic granulomas have more thickened muscular walls and venules as opposed to strictly capillaries. Pyogenic granulomas typically have less inflammation, and the lobules are divided by thickened, fibrous bands.

Another diagnosis to consider is Kaposi's sarcoma. In a Kaposi lesion the vascular spaces are slitlike, and the endothelium is flattened as opposed to the prominent endothelium in this patient. Inflammatory infiltrates associated with Kaposi's lesions tend to contain plasma cells instead of neutrophils. Also, Kaposi's lesions often have red blood cell extravasation with hemosiderin-laden macrophages, which we did not see in this case.
The third major lesion is BA. Classical findings include rounded capillaries, endothelial cell proliferation, neutrophilic infiltrates, and purple granular clusters. Bacillary angiomatosis is caused by *Bartonella henselae* and *Bartonella quintana*. These organisms stain well with silver stains, so we performed a Steiner silver stain that revealed dense, prominent aggregates of bacilli. Thus, the diagnosis of BA was established. We also performed electron microscopy that showed well the extracellular aggregates of coccobacilli, which in this patient were most likely *B. henselae*.

This patient had associated peliosis hepatis and elevated alkaline phosphatase. She also had a history of diarrhea and animal shelter work. She was not homeless and did not have lytic bone lesions or lice infestation. These clinical and epidemiological facts are most consistent with *B. henselae* as opposed to *B. quintana* (32). These 2 organisms can be distinguished by various laboratory methods, including immunofluorescence, colony morphology, polymerase chain reaction, fatty-acid gas-liquid chromatography, and chromogenic enzyme substrates, but, because the very efficacious antibiotic therapy is the same for either organism, these studies are primarily academic (32–35).

**FURTHER DISCUSSION**

DR. MURPHY: Bacillary angiomatosis is a disease characterized by unusual vascular lesions caused by fastidious gram-negative organisms of the genus *Bartonella*. The 2 infecting species are *B. henselae* and *B. quintana*. This infection occurs almost exclusively in patients with HIV infection, although there have been a few case reports of this disease occurring in immunocompetent individuals (32, 36–40).

In 1983, the first case of BA was described in an HIV-infected patient (36). The patient described by Stoeller et al was a 32-year-old man with multiple subcutaneous nodules and a 3-week history of fever, chills, and weight loss. Histopathology of the lesions demonstrated vascular abnormalities and the presence of bacillary forms on Warthin-Starry stain and electron microscopy. At that time, the disease was called *epithelioid angiomatosis* and was believed to be an infection by a previously undefined gram-negative bacillus.

In 1988, an association between BA and cat-scratch disease was proposed (32, 37), and it was thought that these 2 clinical entities may be caused by the same organism in the family *Rickettsiaceae*, namely *Afipia felis*. In 1989, the name of epithelioid angiomatosis was changed to BA (32, 37), and deoxyribonucleic acid analysis proved that *Afipia felis* was not the causative organism of cat-scratch disease or BA, but rather another group of organisms was responsible. In 1992, *Rochalimaea henselae* and *Rochalimaea quintana* were named as causative organisms in these diseases. In 1995, after the development of specific polymerase chain reaction techniques, the organisms were renamed *Bartonella henselae* and *Bartonella quintana*.

Bacillary angiomatosis occurs almost exclusively in HIV-infected patients. Typically, these patients are affected later in the course of their disease. The vascular lesions in the disease usually take 1 of 2 forms. The first form involves the skin, bone, and brain, although many organ systems can be affected. The second form primarily involves the liver and the spleen.
Symptoms at presentation are extremely variable, with severity being related to the significance to which end organs are affected (38, 40). For instance, the clinical spectrum can range from a single cutaneous lesion to overwhelming sepsis. Most patients fall into the former category. The time to presentation also is varied, with patients having skin lesions for >1 year, to this current patient who reported a lesion for only 1 month (39). A history of fever, chills, and weight loss is also a common complaint prior to diagnosis.

The skin lesions are typically cutaneous or subcutaneous and have a wide variety of presentations, including red papules that tend to bleed and large peduncular lesions, plaques, or nodules (39). These lesions are not specific for BA. In fact, differentiating between cutaneous BA and Kaposi's sarcoma is difficult because of the similarity of presentations and images (40). Bone disease often presents as osteomyelitis, typically involving the long bones, and the lytic lesions are positive on bone scans. The bone marrow can be involved with vascular changes, but this is rare (40). In the gastrointestinal tract, the lesions appear with the same variety as the skin manifestations and may cause myriad gastrointestinal symptoms.

When the liver or spleen is involved, the disease is called peliosis hepatis (32, 37). Typically, these patients have relatively normal transaminase levels and a markedly elevated alkaline phosphatase. Additionally, there may be extrinsic compression of the bile duct by the associated lymphadenopathy that may cause a rise in bilirubin levels. Diffuse involvement of the liver with small nodularities also is common. Peliosis of the liver and spleen can occur rarely in patients with advanced cancer, anabolic steroid use, or other medications. All of these abnormalities were seen in this patient, although this was somewhat complicated by the presence of gallstones and the new diagnosis of hepatitis C.

In cases of peliosis, lymphadenopathy often develops in multiple lymph nodes draining affected skin areas, or around the liver, in the case of peliosis. Central nervous system involvement, both as a mass and as meningitis, has been reported, although this too is rare. The bone marrow can be involved with vascular changes, but this is rare.

The differential diagnosis in the current case included benign skin lesion, malignant skin lesion, systemic malignancy, mycobacterial infection, and fungal infection. The skin lesions could be benign lesions, such as dermatofibroma, pyogenic granuloma, angiokeratoma, and cherry angioma (38–40). Possible malignant lesions include Kaposi's sarcoma and angiosarcoma. It is often difficult to distinguish between Kaposi's sarcoma and BA based on skin findings alone. Systemic malignancy may mimic this disease as well as various atypical infections, such as mycobacterial and fungal infections (32, 36–40).

Obtaining tissue for diagnosis is critical. On routine hematoxylin-eosin staining, vascular proliferation and perivascular eosinophilic granular material are seen. The organisms can be identified with a modified silver stain called a Warthin-Starry stain, as well as with electron microscopy. There are enzyme-linked immunosorbent assay methods for detecting antibodies to B. henselae and B. quintana. Both are extremely difficult to culture by either blood or tissue sample. The best culture method involves inoculating both chocolate agar and heart infusion agar with 5% rabbit blood supplement at 35°C (95°F) for at least 3 weeks (40). There are also polymerase chain reaction methods for detecting these organisms (32).
*Bartonella henselae* actually causes 53% of the cases of BA, and the remainder are caused by *B. quintana* (32). The domestic cat, either by scratch or by bite, is the major vector for transmission of *B. henselae* to humans (38, 40). The current patient's exposure to cats at the animal shelter is her likely source of infection.

The human body louse, *Pediculus humanus*, is the principal vector for *B. quintana* infection. *Bartonella quintana* infected thousands in World War I and caused a febrile illness called trench fever. Risk factors for *B. quintana* infection are low income, homelessness, and exposure to lice.

Since its discovery in 1983, BA has been exquisitely sensitive to macrolides (36). An immediate and significant response to macrolide therapy is an important diagnostic tool. Treatment also can include erythromycin, doxycycline, clarithromycin, azithromycin, or ciprofloxacin. Prophylaxis or treatment regimens for MAC that include a macrolide may prevent or treat BA. In a study of 49 patients with BA, no organisms could be cultured once a patient had been given a single dose of a macrolide or a tetracycline (32).

During her hospital stay, our patient had a remarkable response to therapy with a reduction of hepatic enzymes, regression of the umbilical mass, and resolution of hepatic lesions and ascites on repeat sonography. At 1 month after discharge, she was feeling well, and her umbilical mass had completely regressed. Repeat serology showed an HIV-1/RNA titer of >750,000 copies/mL; alkaline phosphatase, 152 U/L; total bilirubin, 2.9 mg/dL; aspartate aminotransferase, 148 U/L; alanine aminotransferase, 119 U/L; lactate dehydrogenase, 37 U/L; and γ-glutamyltransferase, 265 U/L. This case demonstrates a rare manifestation of an AIDS-related illness that responds well to treatment, and, given the recent prevalence of prophylactic regimens containing a macrolide, it may become rarer still.

**References**


