exchange transfusion was curative.[9,11,37,38] Most of these patients were debilitated at the start or had a much higher degree of parasitemia than was seen in the case reported here.

Prevention is based on avoiding ticks.[4] When exposure is unavoidable, frequent body inspections should be done by individual and "buddy" systems. Nymphs should be detached immediately because the tick must feed for at least 12 hours before infection can be transmitted.[4,38] The use of diethyltoluamide-containing insect repellents is also recommended.[38] Unfortunately, transfusion-associated cases will probably continue to occur because widespread screening of donated blood products is not justified based on the low incidence of human infection. Babesiosis is a reportable disease in California (California Code of Regulations, sections 2502 and 2503).

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Rocky Mountain Spotted Fever Following Cardiac Transplantation

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ROCKY MOUNTAIN SPOTTED FEVER was first recognized in the early 1900s in the Snake River Valley of Idaho and the Bitterroot Valley of western Montana.[1] In 1909, Howard Taylor Ricketts established the ixodid tick as the vector for the disease.[2] Following the tick bite, the incubation period averages seven days (4 to 10 days), and the disease varies in severity and course. Typically a patient's temperature remains elevated to 39°C to 40°C (102°F to 104°F). A characteristic rash, a relatively late manifestation, may appear on the wrists and ankles and extend throughout the body, including the palms and soles. Initially there are erythematous macules that blanch with pressure, but after several days the rash becomes maculopapular and petechial. The rash begins to clear as the

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fever resolves, but lesions may remain visible for as long as several weeks. Widespread vascular damage may occur involving the skin, lungs, heart (myocarditis), brain, pancreas, liver, skeletal muscle, or kidneys. The endothelial cell injury leads to increased vascular permeability, edema, hypovolemia, and hypotension.[3]

The etiologic agent, *Rickettsia rickettsii,* is a pleomorphic coccobacillus that is an obligate intracellular parasite. It has ultrastructural similarity to gram-negative bacteria but is poorly visualized by Gram's stain.[4] The organism is more easily detected by direct immunofluorescence of tissue sections from areas of focal inflammation.[5] The primary target cell of rickettsial infection is the vascular endothelial cell. A focal lymphocytic vasculitis is seen on histologic sections of skin lesions. A few neutrophils may be present along with thrombosis and necrosis of the blood vessel. This cellular infiltrate is probably due to destruction of the endothelial cells by the rickettsial organism.[6]

Before the use of antibiotics, Rocky Mountain spotted fever carried a mortality of 15% to 20%.[7] With the use of tetracycline or chloramphenicol, mortality has been lowered to 3% to 6%, although the number of deaths among men, African Americans, and persons older than 40 years remains disproportionately high. Mortality is predictably increased by a delay in either diagnosis or initiation of treatment.[8] Fulminant disease—death within five days of onset—is seen in association with glucose-6-phospate dehydrogenase deficiency.[8] No reports of the spotted fever occurring in an immunosuppressed patient have appeared in the literature, however.

Report of a Case

The patient, a 27-year-old man, underwent orthotopic cardiac transplantation for a congenital mitral valve abnormality. His posttransplantation course was unremarkable, except for several episodes of mild rejection treated with increased immunosuppression. The patient was feeling well before riding for seven days on a cattle drive in southern Utah during the fall season. He traveled in brushy terrain but recalled no tick bites. Four days after his return, he reported myalgias, temperatures to 37.8°C (100°F), and chills. On the second day of the illness, his temperature rose to 40°C with sweats, frontal headache, and sore throat. On the fifth day of the illness, severe arthralgias of his knees, ankles, and metacarpophalangeal joints developed. A nonpruritic rash developed on his wrists and ankles and progressed to involve his palms, soles, and trunk. He did not have cough, earache, dysuria, nausea, or abdominal pain. He had not taken any new drugs except Co-Tylenol, a combination drug containing acetaminophen, chlorpheniramine, pseudoephedrine, and dextromethorphan. He reported several episodes of watery diarrhea without blood or mucus.

The patient was admitted to the University of Utah Medical Center on the fifth day of his illness, at which time his medications included cyclosporine, 180 mg twice a day; prednisone, 5 mg twice a day; captopril, 25 mg twice a day; aspirin, 80 mg a day; and azathioprine, 150 mg a day. He had no known allergies. On physical examination, his blood pressure was 142/70 mm of mercury, pulse rate 104 beats per minute, respirations 32 per minute, and temperature 39°C. The patient appeared cushingoid and diaphoretic with facial flushing, but he was in no acute distress. The cardiovascular examination revealed a prominent fourth sound and tachycardia. No obvious swelling or joint effusions were present in his extremities. Pain was induced by passive or active movement of numerous joints, being most severe in the ankles, knees, wrists, and metacarpophalangeal joints. The skin showed numerous erythematous, nonblanching, maculopapular lesions 2 to 5 mm in diameter scattered on the arms, legs, palms, and soles with a few lesions on his trunk. Several lesions on the lower extremity were petechial.

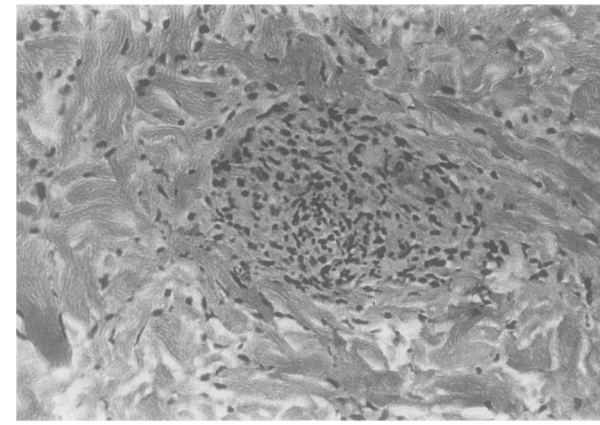

Figure 1.—Histologic examination of skin lesions shows a mononuclear cell infiltrate around the superficial dermal blood vessels (hematoxylin-eosin stain, original magnification × 620). This section was obtained on day 7 of the illness.

The laboratory evaluation revealed microscopic hematuria. Stool specimens were negative for occult blood and leukocytes. Serum electrolyte levels were within normal range. The total bilirubin concentration was 55 μmol per liter (3.2 mg per dl), direct bilirubin 10 μmol per liter (0.6 mg per dl), and the lactate dehydrogenase level was mildly elevated at 433 U per liter. The alanine and aspartate aminotransferase levels were normal. The hematocrit was 0.37 (a month previously it had been 0.41); the leukocyte count was 4.0×10^9 per liter (4,000 per μl) with 0.58 neutrophils, 0.20 band forms, 0.16 lymphocytes, and 0.06 monocytes. Serum analysis for antinuclear antibody was less than 1:40, and a Monospot slide test was negative for infectious mononucleosis. The prothrombin time was 17.4 seconds with an international normalized ratio of 1.2; the partial thromboplastin time was 40 seconds. Serum titers were negative for antistreptolysin-O, Epstein-Barr immunoglobulin (Ig) M, and cytomegalovirus IgM. Rubella and rubeola titers were negative. Biopsy specimens of palpable lesions obtained from the lower extremity on the seventh day of the illness revealed leukocytoclastic vasculitis (Figure 1); periodic acid-Schiff and Gram's stains were negative. A day later a second skin biopsy of a palpable lesion on the left upper extremity was

taken, and sections were stained by indirect immunofluorescence for IgG, IgM, IgA, and C3 with negative results.

On the tenth day of his illness, the patient continued to have chills, severe arthralgias, and a fever to 40°C. Cultures of multiple blood and urine specimens were negative. By that time, the rash was more clearly petechial and had progressed to involve more of his trunk, face, and right conjunctiva. Acute-phase serologic reactions with *R rickettsii* returned negative (less than 1:64). Because of his progressive skin lesions and persistent fever, it was decided to treat the patient empirically for Rocky Mountain spotted fever with doxycycline, 100 mg given intravenously every 12 hours. Within 8 to 12 hours he defervesced, and by hospital day 8 he felt well enough to be discharged on a regimen of oral doxycycline, 100 mg twice a day. At this time the skin lesions had cleared on his trunk, were slowly clearing on his upper extremities, but remained on his lower extremities. His arthralgias had completely resolved.

The current recommendation for antibiotic therapy for Rocky Mountain spotted fever is a seven- to ten-day course[9]; our patient was treated with a three-week course of doxycycline because of his immunosuppression. He did well without a recurrence of skin lesions or symptoms despite the continuation of immunosuppressive therapy. The serologic test at day 70 of the illness was again negative at less than 1:64.

In view of the presumptive diagnosis of Rocky Mountain spotted fever, further staining was done on the biopsy tissue obtained on the tenth day of the illness. This showed typical spotted fever-group rickettsiae using a fluorescein-conjugated polyclonal anti-*R rickettsii* antibody.[10] Further confirmation of the diagnosis was provided when a serum specimen obtained five months after infection had a titer of 1:640 when tested by the indirect fluorescent antibody (IFA) method using *R rickettsii* antigen. In an attempt to exclude possible infection by normally nonpathogenic spotted fever-group rickettsiae, which would be serologically cross-reactive by the direct immunofluorescence method, an indirect immunofluorescence method was adopted using an *R rickettsii*-specific murine monoclonal antibody against a 135-kilodalton species-specific major outer membrane protein of *R rickettsii.* A previously reported method was used that was further modified by the use of a fluorescein-conjugated antimouse immunoglobulin as a second stain.[5,11] The monoclonal antibody did not cross-react with *Rickettsia conorii, Rickettsia australis, Rickettsia akari, Rickettsia montana,* or *Rickettsia sibirica* (J.S.D., unpublished data), and its specificity has been confirmed by Western immunoblot.[12] By this method, *R rickettsii* organisms were visualized in the frozen sections of the skin biopsy, thus strengthening the identity of the specific etiologic agent in this patient.

Discussion

This patient displayed characteristic features of Rocky Mountain spotted fever infection. He presented with fevers and skin lesions typical of a rickettsial disease, and a skin biopsy revealed vasculitis. The presence of rickettsiae within a lesion was shown by immunofluorescent staining for spotted fever group-specific antigens. His response to doxycycline therapy was characteristic of an immunocompetent person with rickettsial infection.

This case is unusual in several respects. Our patient had no recollection of tick exposure; however, less than half of patients with this condition remember the tick bite. Rocky Mountain spotted fever is more frequently seen in the southern Atlantic states; Utah is not an endemic focus. In 1990, the Centers for Disease Control reported 649 cases of the disease, 16 of which were from the western United States and only 3 from Utah.[13] Between 1960 and 1990, there were only 16 cases reported to the State Health Department in Utah. In addition, the disease is more prevalent in the summer months, although this may correlate with tick exposure.

Another unusual aspect of this case is that the patient's serologic tests for Rocky Mountain spotted fever were negative at days 14 of illness and 70 of convalescence. The initial test was done at the University of Utah laboratory using a commercial kit (Hillcrest Biological, Cyprus, California) that detects IgG and IgM antibodies for *R rickettsii* by IFA. Characteristically, the initial IgM class antibody appears by day 8 of primary infection, with IgG detected about day 12. The IFA assay directed against both classes of antibody has been shown to be 95% to 100% sensitive seven to ten days after the illness begins.[14] Furthermore, serologic evidence of previous infection should be detectable beyond 100 days after the onset of the disease.[15] In our case, a serum specimen obtained 150 days after active infection revealed a titer of 1:640 by the IFA method using the rickettsial antigen. We speculate that the delay in mounting an antibody response in this patient was related to the patient's ongoing regimen of prednisone and azathioprine, which are known to dampen immunoglobulin production. In addition, cyclosporine is known to inhibit interleukin-2 production, which limits clonal proliferation of activated cycling B cells and thereby antibody production.[16] Thus, these factors may have contributed to our inability to detect an antibody response earlier in our patient's clinical course.

Recent studies have shown that cell-mediated immunity plays an active role in resistance to rickettsial organisms. Paradoxically, a transient immunosuppression may be seen with clearance of the organism.[17,18] A T-lymphocyte host response including interferon gamma production appears to be important in combating rickettsial infections.[19] Cyclosporine, azathioprine, and prednisone are all potent inhibitors of cell-mediated immunity. The patient presented in this case was taking high doses of cyclosporine and intermediate doses of azathioprine and prednisone. Despite these medications and the absence of detectable antibody during the acute phase, he never became seriously ill. He did have an atypically prolonged course but responded promptly and fully to treatment with appropriate antibiotics. This case suggests that T-cell–mediated mechanisms may be less important than previously thought. Another possible explanation for the unex-

pectedly benign course includes opportunistic infection with a spotted fever-group rickettsial organism that is usually nonpathogenic.

Many nonpathogenic rickettsiae such as *Rickettsia bellii, R montana, Rickettsia rhipicephali,* and *Rickettsia parkeri* have been isolated in the United States.[20,21] They may have antigenic similarities to *R rickettsii* that could produce a false-positive histochemical result in an immunofluorescent test based on the group-specific antigen. We attempted to exclude this possibility by using a murine monoclonal antibody developed against a species-specific major outer membrane protein of *R rickettsii* that is known not to cross-react with *R conorii, R australis, R akari, R montana,* or *R sibirica.* Staining by this technique was positive in our patient. Because this monoclonal antibody has been tested only against one of the nonpathogenic spotted fever-group rickettsiae known to occur in the United States (*R montana*), we are unable to exclude the possibility that the reagent might react with some of the other nonpathogenic tick organisms known to be present in Utah.

Perhaps the exogenous immunosuppression in our patient with a heart transplant provided protection from self-directed attack by the patient's immune system, thereby lessening the endothelial cell damage and resulting vasculitis. Recent investigations with animals suggest that this is not the case. Athymic mice, mice deficient in T cells and B cells, and guinea pigs treated with cyclophosphamide are not protected from immunopathologic responses associated with rickettsiae.[22]

In summary, we have presented a case in which a transplant patient acquired and fully recovered from Rocky Mountain spotted fever despite continuous treatment with potent immunosuppressive medications. Both humoral and cell-mediated immunity are thought necessary to combat rickettsial infections. Unexpectedly, our patient had a benign clinical course and an excellent response to antibiotics despite suppression of both his antibody and cell-mediated immune systems. This suggests that either the patient was infected by nonpathogenic rickettsial species or that other important mechanisms of defense against rickettsial infection exist.

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