

B. microti has been transmitted through blood transfusion in Connecticut (8). Blood collection agencies in southwestern Connecticut and adjacent Westchester County, New York, should be aware of the possibility that blood donors could be infected with this pathogen. Physicians should also be alert to the possibility that patients could be coinfecting with the etiologic agents of Lyme disease or human granulocytic ehrlichiosis. Some patients in whom Lyme disease was diagnosed have been simultaneously infected with *B. microti* (9,10).

Acknowledgments

We thank Michael Vasil and Bonnie Hamid for their technical assistance; the staff of Greenwich Hospital for assistance; Caroline Baisley and Doug Serafin for helping coordinate the trapping of rodents in Greenwich; and Michael Gosciminski, Palma Caron, and Dennis White for providing documentation of several cases of babesiosis from the states of Rhode Island, Massachusetts, and New York, respectively.

This study was supported in part by Hatch funds administered by the U.S. Department of Agriculture.

John F. Anderson* and Louis A. Magnarelli*

*Connecticut Agricultural Experiment Station, New Haven, Connecticut, USA

References

1. Western KA, Benson GD, Gleason NN, Healy GR, Schultz MG. Babesiosis in a Massachusetts resident. *N Engl J Med* 1970;283:854-6.
2. Healy G, Ristic M. Human babesiosis. In: Babesiosis of domestic animals and man. Boca Raton (FL): CRC Press, Inc; 1988. p. 209-25.
3. Centers for Disease Control and Prevention. Babesiosis—Connecticut. *MMWR Morb Mortal Wkly Rep* 1989;38:649-50.
4. Anderson JF, Mintz ED, Gadbaw JJ, Magnarelli LA. *Babesia microti*, human babesiosis, and *Borrelia burgdorferi* in Connecticut. *J Clin Microbiol* 1991; 22:36-8.
5. Herwaldt BL, Springs FE, Roberts PP, Eberhard ML, Case K, Persing DH, et al. Babesiosis in Wisconsin: a potentially fatal disease. *Am J Trop Med Hyg* 1995; 146-51.
6. Herwaldt BL, McGovern PC, Gerwel MP, Easton RM, MacGregor RR. Endemic babesiosis in another eastern state: New Jersey. *Emerg Infect Dis* 2003;9:184-8.
7. Cartter ML, Ertel SH. Lyme disease—Connecticut, 2000, and babesiosis—Connecticut, 1991-2000. Connecticut Epidemiologist Newsletter [serial online] 2001;21:9-12. Available from: [http://www.dph.state.ct.us/Publications/BC H/Infectious%20Diseases/ce72001.pdf](http://www.dph.state.ct.us/Publications/BC%20H/Infectious%20Diseases/ce72001.pdf)
8. Mintz ED, Anderson JF, Cable RG, Hadler JL. Transfusion-transmitted babesiosis: a case report from a new endemic area. *Transfusion* 1991;31:365-8.
9. Krause PJ. Babesiosis. In: The medical clinics of North America: tick-borne diseases. Philadelphia: W. B. Saunders Co.; 2002. p. 361-73.
10. Grunwaldt E, Barbour AG, Benach JL. Simultaneous occurrence of babesiosis and Lyme disease. *N Engl J Med* 1983;308:1166.

Address for correspondence: John F. Anderson, Connecticut Agricultural Experiment Station, PO Box 1106, New Haven, CT 06504, USA; fax: 203-974-8502; email: John.F.Anderson@po.state.ct.us

Migratory Thrombophlebitis and Acute Q Fever

To the Editor: Q fever is a worldwide zoonosis particularly common in some cattle-raising countries such as Great Britain, France, Spain, Australia, and Canada (1). Widespread use of diagnostic serologic tests has led to increasing recognition of the disease in many other parts of the world. For example, Q fever has been recently described in Southeast Asia (2).

Acute Q fever usually manifests as a febrile syndrome with hepatic abnormalities or pneumonia; chronic

Q fever is commonly associated with endocarditis (3,4). When the disease manifests with an atypical clinical picture, diagnosis becomes difficult.

We describe a patient in whom Trousseau's syndrome developed as the sole manifestation of acute Q fever. To the best of our knowledge, this complication has not been found previously in patients with Q fever.

A 39-year-old, previously healthy man was admitted to the hospital because of fever and subcutaneous painful induration in the legs and arms. Ten days previously, he had fever, generalized aches, and malaise. Forty-eight hours after the onset of these symptoms, he noticed a painful induration and redness along the right calf that spontaneously subsided in a few days. He remained febrile, and additional swollen, painful indurated lesions appeared on his thighs and arms. Pleuritic chest pain developed, and he was taken to the emergency room.

On admission, he was febrile (38.7°C). A tender, indurated cord, 10 cm long, was felt on the greater saphenous vein of the right thigh. The cephalic vein of the left arm and the basilic vein of the right arm were also tender and indurated.

The leukocyte count was 9,800/mm³; hemoglobin 14.5 g/dL, and platelets 441,000/mm³. The fibrinogen levels (385 mg/dL) and prothrombin time (12 s, control 10-14 s) were normal. The partial thromboplastin time (40 s, control 26-36 s) was mildly prolonged. Serum aspartate aminotransferase was 75 U/L, serum alanine aminotransferase was 110 U/L, and γ -glutamyltranspeptidase was 230 U/L. Antibodies to phospholipids (aPL) measured by enzyme-linked immunosorbent assay were positive (titer of 95 for antibodies of class IgG/titer of 19 for antibodies of class IgM). Results of testing for tumor-associated antigens, such as carcinoembryonic antigen, carcinoma antigen 19-9, prostatic specific anti-

gen, and alpha fetoprotein, were negative. The x-ray films of the chest and computed tomography scan of the thorax and abdomen were normal. The patient was treated with subcutaneous heparin and diclofenac, and fever and migratory thrombophlebitis subsided.

Because the patient had been working with manure several days before his initial symptoms, Q fever serologic testing was requested. The antibody levels measured by complement fixation (CF) against phase II *Coxiella burnetii* antigen was 1:512. By indirect immunofluorescence, the titers of IgM and IgG against phase I and II were 1:64 and 1:512 and 1:256 and >2,048, respectively. Antibody titers against *Mycoplasma*, *Chlamydia*, *Legionella*, enterovirus, and influenza were negative.

Recovery was uneventful and the patient was asymptomatic during a follow-up visit 3 weeks later. Antiphospholipid antibodies were negative. Three months after the acute phase of the infection, new titers of antibodies (CF) against *C. burnetii* were 1:128. Two years after the episode the patient was asymptomatic.

This patient is unique in that he had acute Q fever with migratory thrombophlebitis. A diagnosis of Trousseau's syndrome associated with an occult malignancy was considered on admission, but it was excluded soon. The recent history of exposure to manure was the key for the clinical diagnosis. Although specific anti-coxiella treatment was not given, the patient followed a self-limited course, and both clinical and laboratory abnormalities promptly subsided.

Microscopic vasculitis and thrombosis are commonly found in patients with other rickettsial infections (5), but vascular phenomena must be considered an exceptional event in patients with Q fever. However, thrombophlebitis and pulmonary embolisms have been occasionally reported (6–8). These unusual mani-

festations have been associated with aPL during the course of acute Q fever (7,8).

Antibodies to phospholipids have been found in 80% of patients in a large series of acute Q fever (9). None of the patients in the study showed thrombotic events or cardiac valve involvement in contrast to patients with lupus or primary aPL syndrome in whom clinical manifestations attributed to aPL developed (9). This observation could be explained by the fact that aPL found in patients with lupus and primary aPL syndrome are directed against a complex antigen that includes a lipid-binding inhibitor of coagulation, which has been characterized as a β 2-glycoprotein I (apolipoprotein H). This glycoprotein seems to inhibit the activation of the contact phase system of the intrinsic pathway of blood coagulation (10). On the other hand, apolipoprotein H is not necessary for the aPL activity observed in patients with Q fever and other infectious diseases (10).

According to these studies, the observation of low titers of aPL in the serum of our patient during the acute phase of Q fever must be seen as a finding of uncertain importance not necessarily associated with migratory thrombophlebitis. In short, migratory thrombophlebitis (Trousseau's syndrome) should be added to the ever-growing list of unusual manifestations of Q fever.

**Manuel L. Fernández Guerrero,*
Pablo Rivas,*
and Rosa García Delgado***

*Fundación Jiménez Díaz, Madrid, Spain

References

1. Reimer LG. Q fever. *Clin Microbiol Rev* 1993;6:193–8.
2. Suputtamongkol Y, Rolain JM, Losuwanaruk K, Niwatayakul K, Suttinont C, Chierakul W, et al. Q fever in Thailand. *Emerg Infect Dis* 2003;9:1186–8.
3. Raoult D, Tissot-Dupont H, Foucault C, Gouvenet J, Fournier PE, Bernit E et al. Q fever 1985 to 1998. Clinical and epidemiologic features of 1,383 infections. *Medicine (Baltimore)* 2000;79:109–23.
4. Hatchette TF, Marrie TJ. Atypical manifestations of chronic Q fever. *Clin Infect Dis* 2001;33:1347–51.
5. Elghetany MT, Walker DH. Hemostatic changes in Rocky Mountain spotted fever and Mediterranean spotted fever. *Am J Clin Pathol* 1999;112:159–68.
6. Cuenca A, Simeón CP, Menieve A, Bosch GM, Gil JA, Ordi Ros J. Fiebre Q y tromboembolismo pulmonar. *Rev Clin Esp* 1990;187:151–2.
7. Gálvez J, Martín I, Merino D, Pujol E. Tromboflebitis e un paciente con fiebre Q aguda y anticuerpos anticardiolipina. *Med Clin (Barc)* 1997;108:396–7.
8. Del Arco A, De la Torre J, Prada JL, García J. Tromboembolismo pulmonar en paciente con fiebre Q ¿ son los anticuerpos anticardiolipina la causa? *Enferm Infecc Microbiol Clin* 2001;19:137–8.
9. Ordi Ros J, Selva A, Monegal F, Monasterio Y, Juste C, Vilardell M. Prevalence, significance, and specificity of antibodies to phospholipids in Q fever. *Clin Infect Dis* 1994;18:213–8.
10. McNeil HP, Simpson RJ, Chesterman CN, Krillis SA. Anti-phospholipids antibodies are directed against a complex antigen that includes a lipid-binding inhibitor of coagulation: β 2-glycoprotein I (apolipoprotein H). *Proc Natl Acad Sci U S A* 1990;87:4120–4.

Address for correspondence: Manuel L. Fernández Guerrero, Division of Infectious Diseases, Fundación Jiménez Díaz, Avda. Reyes Católicos, 2, 28040 Madrid, Spain; fax: 34-91 550 49 22; email: mlfernandez@fjd.es

West Nile Poliomyelitis

To the Editor: In the July 2003 article, "Acute Flaccid Paralysis and West Nile Virus Infection" (1), Sejvar et al. reported seven patients with acute onset of asymmetric weakness and areflexia but no sensory abnormalities. The authors also referenced three previously reported cases of West Nile virus (WNV)-associated flaccid paralysis and argued that all of these symptoms could be explained by anterior-horn cell loss. The two