

in transmission.

The clinical manifestations of Lyme disease occur in three different stages. The hallmark of Stage I is erythema chronicum migrans (ECM), which presents in 80% of patients 4 to 32 days after the tick bite (only one-third of patients with ECM will remember a tick bite). ECM begins as papular lesion at the site of the tick bite. The lesion may enlarge to as much as 68 cm in diameter, with central clearing and a rim of erythema. Multiple satellite lesions occur in about half of patients; these lesions are usually smaller, without central clearing.

Treatment with tetracycline, 250 mg qid for 10 days, will usually prevent progression to Stages II and III disease. Children and pregnant women are treated with penicillin or, in the case of penicillin allergy, erythromycin.

Constitutional symptoms may be the only manifestations of the disease in 20% of patients. Diagnosis is difficult because the symptoms mimic those of a variety of infectious and noninfectious diseases. The patient who seeks medical attention may complain of malaise, fatigue, lethargy, headache, fever, and chills. Myalgias, backache, sore throat, and diarrhea also may occur.

Stage II disease consists of cardiac or neurologic involvement, and develops in 8 to 15% of patients who are untreated in Stage I. Cardiac involvement, including fluctuating degrees of atrioventricular block and myocarditis with left ventricular dysfunction, is usually brief, self-limited, and treated symptomatically. Pacing may be required if complete heart block develops, but cardiac symptoms usually resolve within a few days to six weeks.

Neurologic manifestations develop in 10 to 15% of patients who are not treated during Stage I. The classic clinical presentation is a triad of aseptic meningitis, cranial nerve palsies, and peripheral radiculoneuropathy, although these may occur separately. Waxing and waning cranial neuropathies are an important clinical clue to the diagnosis. Neurologic manifestations are treated with high-dose parenteral penicillin; data are inadequate to assess the efficacy of oral penicillin.

Relapsing or remitting chronic arthritis, most commonly involving the knee, is the major manifestation of Stage III Lyme disease. Chronic Lyme arthritis is treated with high-dose parenteral penicillin. Ceftriaxone, 2 or 4 g/d, may become the agent of choice for arthritis patients whose disease is unresponsive to penicillin (There are no data on the use of ceftriaxone for Stage I disease, although it is being evaluated in an ongoing study.)

Diagnosis of Lyme disease at all three stages depends heavily on clinical observation. Biopsy is generally insensitive and culture is cumbersome and usually not commercially available. Serologic diagnosis is notoriously unreliable for diagnosis during Stage I disease; serologic studies are positive in only 10% of patients who present with ECM. Although enzyme-linked immunosorbent assay and indirect fluorescence assay have been touted for their sensitivity in Stages II and III disease, recent data suggest problems with specificity, particularly in the setting of lupus erythematosus, rheumatoid arthritis, and other spirochetal diseases.

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BIBLIOGRAPHY

Steichenberg BW. Lyme disease: the latest great imitator. *Pediatr Infect Dis* 1988; 7:402-409.

Barbour AG. Laboratory aspects of Lyme borreliosis. *Clin Micro Rev* 1988; 1:399-414.

CLUES TO DIAGNOSIS OF A LIFE-THREATENING THROMBOTIC SYNDROME

Early diagnosis and aggressive management of Trousseau's syndrome is essential for patient survival. The syndrome is characterized by venous or arterial thrombotic events associated with occult, often metastatic tumors; the neoplasms commonly involve the lung, pancreas, stomach, colon, or prostate. The thrombotic event can precede diagnosis of the malignancy by as much as one year.

Diagnosis requires a high index of suspicion. Thrombotic events that suggest underlying malignancy include migratory superficial thrombophlebitis, upper extremity thrombosis, multiple-site involvement, and highly inflammatory phlebitis. Thrombosis resistant to anticoagulant therapy also is suggestive.

Trousseau's syndrome should be considered when thrombotic events or pulmonary embolism occur in the absence of predisposing factors such as immobilization, pregnancy, or recent orthopedic or other surgery.

CAREFUL WORKUP

With a suspicious presentation, the recommendation is to complete a careful history and physical examina-

tion. The laboratory workup should include complete blood count, peripheral blood smear, urinalysis, chest radiograph, and stool for occult blood. A female patient should have a pelvic examination and Pap smear, and mammography. Further workup may include CT scans and MRI scans.

Laboratory features that suggest Trousseau's include hypofibrinogenemia and thrombocytopenia, but laboratory findings may be normal. Other findings include elevation in fibrin degradation products, prolonged prothrombin time, or activated partial thromboplastin time. Microangiopathic hemolytic anemia in the setting of a clotting disorder and weight loss suggests underlying malignancy.

Identification and aggressive treatment of the tumor with surgery, chemotherapy or radiation is essential. Without partial or complete response of the tumor, the prognosis for survival is approximately 3 to 4 months

after the diagnosis of Trousseau's has been established.

Anticoagulation is helpful. Although few patients respond to coumadin, treatment with heparin usually is beneficial and must be continued indefinitely.

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BIBLIOGRAPHY

Bell WR, Starksen NF, Tong S, Porterfield JK. Trousseau's syndrome: devastating coagulopathy in the absence of heparin. *Am J Med* 1985; 79:423-430.

Rickles FR, Edwards RL. Activation of blood coagulation in cancer: Trousseau's syndrome revisited. *Blood* 1983; 62:14-31.

Sack Jr GH, Levin J, Bell WR. Trousseau's syndrome and other manifestations of chronic disseminated coagulopathy in patients with neoplasms: clinical, pathophysiologic, and therapeutic features. *Medicine* 1977; 56:1-37.

ERRATA

The organism *Campylobacter pylori* was spelled incorrectly as *C pyloris* on pages 12-13 of the January/February 1989 (volume 56) issue.

On page 206 of the March/April 1989 (volume 56) issue, in the article, "Pulmonary infiltrates and eosinophilia revisited," by David P. Meeker, MD, the section referring to a dosage of 5 mg/kg/day prednisone should have stated 0.5 mg/kg/day.