tuted CD4+ T-cell subset was involved in promoting the occurrence of this HIV-polymyositis.

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TRANSIENT ANTICARDIOLIPIN ANTIBODY SYNDROME IN A PATIENT WITH PARVOVIRUS B19 INFECTION

To the Editor:

Transient autoantibodies are often produced in response to viral infections (1,2). Although these autoantibodies are usually polyspecific and occur in low titers, some patients develop high-titer antibodies with subtype specificity.

Human parvovirus B19 causes several clinical syndromes including erythema infectiosum, polyarthritis, hepatitis, autoantibody expression, and aplastic crises in patients with hemolytic anemia or hemoglobinopathies (3,4). Anticardiolipin antibodies have been reported infrequently in adults infected with parvovirus B19. We report a patient with transient anticardiolipin antibody syndrome and thrombosis after human parvovirus B19 infection.

A 25-year-old previously healthy man was admitted with left upper-quadrant abdominal pain and a 2-week history of fever. Physical examination revealed a fever of 38.8°C and tender splenomegaly. There was no hepatomegaly or lymphadenopathy, and no other abnormalities were noted.

Splenomegaly (and the absence of hepatomegaly) were confirmed with an abdominal ultrasonography examination. Persistent abdominal pain and worsening splenomegaly prompted a second ultrasound examination with duplex, which revealed a wedge-shaped lesion in the spleen consistent with an infarct and reversed flow in one of the branches of the splenic vein. There was no evidence of thrombosis in the hepatic vein or the main splenic vein. A computed tomographic scan of the abdomen confirmed the infarct area in the spleen and showed mild retroperitoneal lymphadenopathy.

Laboratory studies disclosed normal levels of antithrombin-III, protein C and protein C resistance, protein S, and homocysteine, and elevated titers of anticardiolipin antibody IgG (104 gpl/mL) and IgM (56 mpl/mL). Two weeks after presentation, further tests were strongly positive for IgM antibodies and slightly positive for IgG antibodies for parvovirus B19. There was no evidence of IgM antibodies for cytomegalovirus or Epstein-Barr virus infections.

The patient's condition was diagnosed as acute parvovirus B19 infection with the anticardiolipin antibody syndrome. He was treated with aspirin (100 mg per day) and his fever and upper abdominal pain resolved. An abdominal ultrasonography examination revealed normal flow in the splenic vein. Repeat tests 1 and 2 months later showed decreases in the titers of IgG and IgM anticardiolipin antibodies.

The natural history of human parvovirus B19 infection is biphasic, consisting of a viremic phase and an antibody response phase (3). Some patients are asymptomatic; others have a flulike illnesses. The onset of anti-B19 IgM antibodies is associated with clearance of viremia, and the second phase of the illness is frequently characterized by rash, arthralgia, or arthritis, and less frequently by autoantibodies in the blood (3,5,6).

Moore et al (7) reported 7 patients with parvovirus B19 infection who had a history of malar rash and arthralgia. Six of these patients had antinuclear antibodies, of whom 2 had antibodies to Scl-70, and 4 had antibodies to Sm, RNP, SS-A (Ro), or SS-B (La). Two patients presented with elevated levels of rheumatoid factor, and all had an elevated IgM antibody titer to parvovirus B19 at the onset of their illness. Hansen et al (1) reported a 54-year-old woman who presented with a history of febrile disease and diffuse pruritic rash. Laboratory studies showed evidence of anticardiolipin IgG and IgM. Further investigation revealed IgM antibodies to parvovirus B19.

Anticardiolipin antibodies have been found in a variety of viral illnesses. In one study (8), 40 patients with acute viral infection were tested for anticardiolipin antibodies by enzyme-linked immunosorbent assay. Eight of 10 patients with parvovirus B19 infection, 7 of 10 with Epstein-Barr virus infection, 10 of 10 with hepatitis A virus infection, and 8 of 10 with rubella had anticardiolipin antibodies.

Infection with parvovirus B19 may be associated with the expression of various autoantibodies and clinical events, as in our patient. In patients with prolonged febrile illness and

symptoms of connective tissue disease, testing for parvovirus B19 antibodies may be helpful in differentiating between transient illness and connective tissue disease and in making decisions about patient management.

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LACTOCOCCUS GARVIEAE SEPTICEMIA WITH LIVER ABSCESS IN AN IMMUNOSUPPRESSED PATIENT

To the Editor:

Lactococcus garvieae was first isolated from cases of bovine mastitis, and it may also infect several species of fish (1–3). Infection in humans is rare (4–6).

A 68-year-old woman was admitted to our intensive care unit with gastrointestinal bleeding. Two months before admission, a cholangiocarcinoma involving the intrahepatic branches of the biliary tract had been discovered. A Teflon biliary prosthesis was placed in each of the branches, and treatment with prednisone was begun. No recent history of infection or contact with fish or animals was reported.

On admission, the patient had no abdominal pain, fever, or chills. Physical examination showed a cachectic, pale, and icteric woman. Her temperature was 37.1°C. Other clinical findings were normal. Laboratory studies showed a hemoglobin level of 6.6 g/dL, a hematocrit of 26%, a white blood cell count of 28,900/μL with 86.6% neutrophils, a total bilirubin level of 121 μM, an aspartate aminotransferase level of 181 IU/L, and a lactate dehydrogenase level of 694 IU/L. Gastrointestinal endoscopy revealed active bleeding in the second portion of the duodenum, which suggested hemobilia.

The patient received a blood transfusion, and treatment with omeprazole was begun. A retrograde cholangiography showed obstruction of the two prostheses and an intrahepatic cavity (4 cm in diameter) that communicated with the left intrahepatic branch of the biliary tract, suggesting abscess. Three blood cultures obtained at admission grew streptococci. The prostheses were removed and treatment with amoxicillin (1 g three times daily), netilmicin (250 mg daily), and metronidazole (500 mg three times daily) was begun. The organism was identified as *L. garvieae* using a rapid system for identification of streptococci and related organisms (rapid ID 32 STREP; bioMérieux sa, Marcy-l'Etoile, France), which consists of 32 biochemical reactions that are read after an incubation period of 4 hours. The organism was susceptible to amoxicillin, tetracycline, erythromycin, vancomycin, and teicoplanin. The patient died on the 12th hospital day from a massive gastrointestinal hemorrhage.

There have been only a few reports of human infection with lactococci (5,7,8). However, because it is difficult for many microbiology laboratories to distinguish between lactococci and enterococci (5), human infections may be underdiagnosed. In addition, the published biochemical and antigenic characteristics of *L. garvieae* do not differentiate this pathogen from *L. lactis* (2). A specific polymerase chain reaction assay or antibiotic susceptibility tests may help distinguish the two pathogens (2,4,5). Indeed, *L. garvieae* seems to be more resistant to clindamycin than is *L. lactis* (2,4).

Although *L. garvieae* could not be cultured from the infected bile in our patient, we believe that it was the causal agent of the liver abscess, and this collection was the primary source of bacteremia.

Prior reports have identified approximately 15 strains from clinical specimens, most of which were cultured from blood (4,6). In 3 cases, the pathogen involved prosthetic valves (5). The significance of the other isolates was less clear (6). In 1998, a case of septicemia and native valve endocarditis was reported (5). As in our case, the patient was immunosuppressed, suggesting the possible invasiveness of this pathogen in these patients. Physicians should be aware of the possible severity of the disease and institute prompt antibiotic treatment, particularly in immunosuppressed patients.

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