in the nasopharynx sets the stage for sinusitis/otitis more frequently than that. The seasonal incidence of otitis media and streptococcal pharyngitis are similar.

ETD is generally acknowledged as a precursor to otitis media. It would be important to know if infection and/or asymptomatic colonization of the nasopharynx with GABHS is an etiological factor in otitis media.

The results of this survey suggest that an evaluation of the incidence of GABHS in the nasopharynx of children with otitis media may be an important issue in our understanding of middle ear disease.

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LYME DISEASE PRESENTING AS HEPATITIS AND JAUNDICE IN A CHILD

Lyme disease is a tick-borne disease caused by *Borrelia burgdorferi*. Its major manifestations are rheumatic, dermatologic, neurologic and cardiac. Less frequently other organ systems including the liver and gastrointestinal tract are involved.

In a series of 314 patients with erythema chronicum migrans which included some children as young as 2 years of age, Steere et al.[1] reported that 10% had symptoms suggestive of hepatitis, 5% had hepatomegaly and 19% had elevated aspartate aminotransferase concentrations with a median value of 71 units/liter (range of 36 to 251). Reports of an adult with chronic hepatitis resulting from recurrent Lyme disease[2] and an adult with granulomatous hepatitis associated with Lyme disease[3] have appeared. Furthermore pathologic specimens of liver from patients of unspecified age in the early stage of Lyme disease have shown lesions ranging from mild lymphocytic portal triaditis to liver cell derangement resembling acute viral hepatitis.[4] *B. burgdorferi* has been demonstrated in small numbers in pathologic specimens of liver.[5]

In Westchester County, NY, an area highly endemic for Lyme disease, 40% of reported cases of Lyme disease were in children less than the age of 19.[6] The highest national age-specific incidence rate among 6900 cases reported to the Centers for Disease Control in 1987 and 1988 was in children less than 15 years of age.[7] Although reviews of Lyme disease in children mention hepatitis,[8, 9] reference is made only to adult cases. There has not, to our knowledge, been a report of hepatitis as the presenting manifestation of Lyme disease in a child.

Case report. In July, 1989, a 6-year-old boy residing in Westchester County, NY, presented for evaluation of jaundice and a limp. He complained of fever to 103°F for 6 days, associated with several episodes of nonbilious vomiting and an evanescent "prickly-heat" truncal rash. He had had anorexia for 5 days, pain in his left knee and thigh for 3 days and a limp for 2 days. Forty-eight hours before presentation the patient developed loose stools which resolved after 24 hours. On the day before presentation his mother noticed that his eyes appeared yellow.

He had no history of a tick bite. Deer were not seen in his neighborhood, but he often accompanied his father on fishing trips to wooded areas in northern Westchester County. The child had not recently received any medications and had never received a blood transfusion. Two weeks before onset of the child's illness, his father had been diagnosed to have serologically positive Lyme disease after 4 weeks of arthralgia and fatigue. The father had no symptoms or signs of hepatitis.

Physical examination revealed a moderately ill-appearing boy with a temperature of 103°F and a macular erythematous rash on the thighs. He had icteric sclera. Chest and cardiac examinations were normal. The abdomen was soft with normal bowel sounds and was diffusely tender without rebound. A smooth, mildly tender liver edge was palpated 3 cm below the right costal margin. The spleen was not palpable. There were no other abdominal masses. There was no swelling, warmth or redness of joints, but there was mild pain on abduction and adduction of the left hip.

Laboratory results included a white blood cell count of 10,200 cells/mm^3 with 67% neutrophils, 19% lymphocytes, 2% monocytes and 2% eosinophils; hemoglobin 11.3 g/dl; erythrocyte sedimentation rate 94 mm/hour; prothrombin time 11.8 seconds (normal, 11.5 seconds), partial thromboplastin time 26.9 seconds (normal, 26.7 seconds); aspartate

aminotransferase 60 units/liter (normal 0 to 37), alanine aminotransferase 80 units/liter (normal 0 to 40), gamma-glutamyltransferase 118 units/liter (normal 7 to 50), total bilirubin 3.1 mg/dl, direct bilirubin 2.3 mg/dl, albumin 3.7 g/dl and total protein 6.7 g/dl. Serological studies for hepatitis B surface antigen, hepatitis A antibody, Epstein-Barr virus, antinuclear antibody and rheumatoid factor were negative. Lyme titer was 419 by a quantitative indirect immunofluorescence assay (FIAX; Whittaker; the upper limit of negative was 50; values of greater than 75 were considered positive) and was 66 units by enzyme-linked immunosorbent assay using polyvalent IgG and IgM antiserum (The upper limit of negative was 14. Values of greater than 20 were considered positive). An abdominal radiograph suggested a questionably calcified retroperitoneal mass.

However, an ultrasound examination of the abdomen could not confirm the presence of calcification or mass and showed normal liver architecture and hepatic vessels. A computed tomography scan of the abdomen confirmed the ultrasonographic findings. Radiographs of the hips and knees were normal.

The patient was treated with 250 mg of amoxicillin tid orally for 21 days. By Day 3 of treatment he was afebrile, more active and less icteric. On reevaluation 10 days after the first visit he appeared well and the leg pain had resolved. Aspartate aminotransferase, alanine aminotransferase, gamma-glutamyltransferase and bilirubin concentrations were normal. Serologic studies for cytomegalovirus (IgG and IgM), *Leptospira*, hepatitis B surface antigen and anti-hepatitis B surface antibody were negative. He remains asymptomatic 4 months after his diagnosis.

Discussion. We have described a child living in an area endemic for Lyme disease who presented during the season of highest incidence of Lyme disease with jaundice, fever, hip pain, and elevated serum transaminase values and direct hyperbilirubinemia, suggestive of acute viral hepatitis. Investigation revealed an increased antibody titer against *B. burgdorferi* and no evidence of any other diagnosis to explain these findings.

While it is not uncommon in our experience for mildly elevated liver function tests and gastrointestinal complaints to be present in children with Lyme disease (M Glassman, KS Edwards, KI Li, S Kanengiser, unpublished data), we have not previously seen children with hepatitis and jaundice as the initial manifestation of the disease, and could not identify other reports in the literature of acute-onset hepatitis as the presenting manifestation of Lyme disease in children.

Given the high incidence of Lyme disease in Westchester County, the occurrence of asymptomatic seroconversion, and evidence that specific antibodies may persist, it was important to consider other etiologies of hepatitis in this child even though the serologic tests for Lyme disease were markedly positive. Other causes of acute hepatitis include toxic drug effects, viral infection, Wilson's disease, alpha 1-antitrypsin deficiency, ischemia and Reye's syndrome. In view of the absence of a history of drug or toxin ingestion or transfusion, a physical examination revealing no evidence of Wilson's disease or hypotension, and his rapid response to antibiotic therapy without subsequent recurrence of symptoms, other etiologies of this patient's liver disease were unlikely. Although the positive serological tests do not prove that *B. burgdorferi* was the causative agent in this case, *B. burgdorferi* infection is associated with hepatic dysfunction and other known causes of hepatitis here excluded.

As pediatricians practicing in endemic areas are learning, Lyme disease is truly the latest "great imitator,"[8] and the diagnosis must be considered in the differential diagnosis for an increasingly broad spectrum of problems. Lyme disease should be considered among the infectious causes of acute hepatitis in children who live in or vacation in endemic areas.

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PARVOVIRUS-ASSOCIATED APLASTIC CRISIS IN A PATIENT WITH RED BLOOD CELL GLUCOSE-6-PHOSPHATE DEHYDROGENASE DEFICIENCY

Human parvovirus B19 (HPV) is the etiologic agent of erythema infectiosum.[1] This virus has also been associated with aplastic crises in patients with underlying chronic hemolytic anemias, including sickle cell disease,[2] hereditary spherocytosis,[3] pyruvate kinase deficiency[4] and thalassemia.[5] In this report we describe a child with glucose-6-phosphate dehydrogenase (G-6-PD) deficiency who developed a profound anemia, reticulocytopenia and mild leukopenia during an acute parvoviral infection. This is the first report of HPV-associated aplastic crisis in G-6-PD deficiency.

Case report. A 13-year-old Caucasian male with known G-6-PD deficiency presented to the emergency room with a 3-day history of malaise, fever, headaches and increased