

Bile Duct Abnormalities in the Acquired Immune Deficiency Syndrome

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Pathological changes in the liver have been described in patients with acquired immune deficiency syndrome; however, lesions of the bile ducts have not been noted. We report 2 patients with intrahepatic bile duct abnormalities, one having sclerosing cholangitis of the large ducts. We discuss the possibility that these changes are secondary to opportunistic infection or are induced more directly by immunologic mechanisms.

Relatively little has been published regarding hepatic involvement in patients with acquired immune deficiency syndrome (AIDS) (1-3). The histopathologic changes noted in the liver at autopsy of patients with AIDS include sinusoidal dilatation with adjacent atrophy of liver cells, binucleated hepatocytes, fatty changes, widened portal tracts with some fibrosis, bile duct proliferation, and granulomas and tumors (4).

Cholestatic liver function tests have been reported in patients with AIDS in association with intrahepatic granulomas; however, no lesions of either intrahepatic or extrahepatic bile ducts have been documented. We report 2 cases of AIDS with elevation of the alkaline phosphatase, intrahepatic bile duct abnormalities, and in 1 case, sclerosing cholangitis.

Case Reports

Case 1

A 43-yr-old homosexual man was admitted to Memorial City General Hospital, Houston, in February 1986

because of diarrhea and fever. In November 1984 the patient developed watery, bloodless diarrhea, up to 20 bowel movements per day, not associated with abdominal pain or weight loss. Three months later, because of persistent symptoms, he was investigated with upper GI series, small bowel examination, barium enema, gastroscopic examination, colonoscopy, and bronchoscopy; all were within normal limits. All laboratory studies were normal. In April 1985 the patient continued to have diarrhea and had developed fever and night sweats. Two months later he became afebrile following antibiotic therapy for a suspected sinus infection. In December 1985 the patient had recurrent fever, night sweats, epigastric pain, and increasing diarrhea, at which time he was diagnosed to have AIDS and cryptosporidium infection.

He had a history of hepatitis in 1981 and had been drinking half a pint of vodka per day until 2 yr before admission when he decreased alcohol consumption to an occasional drink. The patient had used nonparenteral illicit drugs and had been receiving thiothixene, 2 mg daily, for 2 yr. On physical examination, the patient was found to be cachectic and had oral candidiasis; no peripheral adenopathy was noted. The abdomen was soft and tender to palpation at the epigastrium. He had diffuse hepatomegaly with a 15-cm span at midclavicular line. Rectal ulcers were diagnosed as herpes virus infection.

Laboratory studies demonstrated a hemoglobin of 10.5 g, hematocrit of 32%, and a white blood cell count of 5500/mm³ with 71 neutrophils, 10 bands, and 17 lymphocytes. Alkaline phosphatase was 415 U/dl (normal, 0-115 IU), serum glutamic oxaloacetic transaminase was 43 U/dl, and albumin was 3.5 g/dl. The HTLV-III was positive and stools were negative for cryptosporidium and leukocytes. Immunoglobulin A was elevated at 435 mg/dl, but immunoglobulin G and immunoglobulin M were normal. The T-helper lymphocyte level was 55/mm³ and the T-suppressor level was 382/mm³ with a reversed ratio of 0.1. Hepatitis B surface antigen was negative, hepatitis B surface B antibody was positive, hepatitis B core antibody was positive, and hepatitis A virus antibody was negative. A computed axial tomography scan of the abdomen re-

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Abbreviations used in this paper: AIDS, acquired immune deficiency syndrome; CMV, cytomegalovirus.



Figure 1. Cholangiogram consistent with sclerosing cholangitis.

vealed bile duct dilatation. At this time endoscopic retrograde cholangiopancreatography was done and revealed changes consistent with sclerosing cholangitis (Figure 1).

At the time of endoscopic examination, the ampulla of

Vater appeared to be friable and edematous. Biopsy specimens from this area revealed the presence of cytomegalovirus (CMV) inclusion bodies in the nuclei of cells lining periductal glands (Figure 2). A needle biopsy of the liver demonstrated, in addition to sinusoidal congestion, bile duct abnormalities in the portal tracts (Figures 2 and 3). These findings included distortion of the interlobular ducts by coarse bundles of fibrous tissue, swollen duct epithelial cells with vacuolated enlarged reactive nuclei, and in some ducts focal loss of epithelium. In a few portal tracts there was complete absence of the bile duct. Proliferation of ductules at the margins of the portal tracts was present but not prominent. Most portal tracts demonstrated a mild lymphocytic infiltrate, and in some of the abnormal bile ducts there were lymphocytes within the bile duct epithelium. A few portal tracts contained normal interlobular ducts.

The patient followed a protracted course manifested by intermittent diarrhea, progressive weight loss, and subsequent confusion and lethargy. Two months after admission he died.

Case 2

A 42-yr-old male homosexual was admitted to Memorial City General Hospital in June 1984 for evaluation of epigastric pain. There was no history of fever, sweating, or diarrhea. He denied hepatitis, venereal disease, alcohol consumption, and the use of illicit drugs.

Upon examination the abdomen was soft but tender at



Figure 2. Taken from ampulla of Vater: enlarged cell (arrow) with typical CMV nuclear inclusion. (H&E stain, $\times 400$.)

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