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STRATEGIES FOR THE TREATMENT OF AIDS-ASSOCIATED SCLEROSING CHOLANGITIS

To the Editor:

Acquired immunodeficiency syndrome (AIDS) is occasionally associated with abnormalities of the biliary tract (1). For one of these abnormalities, AIDS-associated sclerosing cholangitis, there are no effective therapeutic measures available (2,3).

We studied 9 patients who had human immunodeficiency virus (HIV) and AIDS-associated cholangiopathy who were selected on the basis of clinical manifestations, serum levels of alkaline phosphatase, and ultrasonographic abnormalities. Of these patients, 6 were men and 3 were women,

with a mean age of 29 years (range, 21 to 56 years). Eight patients had right upper quadrant pain and diarrhea, and 1 patient was asymptomatic.

The most marked abnormality on routine hepatic biochemical testing was an increased serum alkaline phosphatase level (mean, 1200 U/L; range 500 to 3000 U/L). CD4 lymphocyte counts were <100/µl.

The ultrasonographic abnormalities were considered when images demonstrated periportal echogenic bands, common bile ducts greater than 8 mm in diameter, thickening of common bile ducts with or without dilatation, and intrahepatic bile duct dilation (4,5). Ultrasonography in 8 patients showed bile duct dilation. Five patients had dilation and thickening of bile ducts. One patient had thickening but not dilatation of the bile duct.

All patients were studied by endoscopic retrograde cholangiography (6) with papillary biopsy or magnetic resonance cholangiography (7). Endoscopic retrograde cholangiography or magnetic resonance cholangiography showed sclerosing cholangitis in 7 patients and sclerosing cholangitis and papillary stenosis in 2 patients.

Stool samples obtained from all patients were examined for parasites, including coccidian oocysts and microsporidia (8). Papillary and duodenal biopsy specimens were obtained for routine histology and transmission electron microscopy to confirm the species of microsporidia (8). These studies revealed pathogens in 7 patients. In patients with sclerosing cholangitis the diagnosis was cryptosporidiosis in 3, isosporosis in 1, and cryptosporidiosis associated with microsporidiosis in 1 patient. In patients with sclerosing cholangitis and papillary stenosis the diagnosis was microsporidiosis in 2 patients. In the 3 patients with a diagnosis of microsporidiosis, the organism was confirmed as Enterocytozoon bieneusi.

Five patients were not treated, as they had negative results for parasites or *Cryptosporidium* sp. infection. Two patients were treated for microsporidia only (9), and another 2 patients received antiretroviral therapy in combination with the treatment for each specific pathogen (microsporidia and *Isospora belli*). Endoscopic sphincterotomies were considered in 2 patients with papillary stenosis (10), one of whom was treated with albendazole alone and the other with albendazole and highly active antiretroviral therapy (HAART).

Subjects were followed for 3 to 22 months. Five patients without HAART and specific pathogen treat-

Table. Characteristics of Patients with AIDS-associated Cholangiopathy who Received HAART during Follow-up

Characteristic	Patient 3	Patient 9
HAART	AZT, 3TC, IDV	AZT, 3TC, NFV
Resolution of diarrhea (week)	8	6
Resolution of pain (week)	4	3
Follow-up (month)	22	10
Alkaline phosphatase level (mean)	2000 U/L to normal	1000 U/L to normal
CD4 lymphocyte count (cells/ μ l)	7 to 100 cells/μl	79 to 180
Serum viral load (copies/mL)	29,510*	168,471 to 3705
Ultrasonography	Extrahepatic bile duct dilation with distal stenosis. Dilation reduced from 11 to 9 mm	Extrahepatic bile duct dilation that reduced from 11 to 8 mm.
Magnetic resonance cholangiography	Intrahepatic bile ducts dilation. Extrahepatic bile duct dilation of 11 mm with distal stenosis*.	Intrahepatic bile ducts from dilated to normal. Extrahepatic bile duct dilation that reduced from 12 to 9 mm.
Relapse (months)	Yes, diarrhea (22)	No

³TC = lamivudine; AZT = azidothymidine; HAART = highly active antiretroviral therapy; IDV = indinavir; NFV = nelfinavir; ZDV = zidovudine.

^{*} Not controlled.

ment survived for 4 to 20 months or were still alive after completion of the study. One patient treated only for specific pathogens and endoscopic sphincterotomy died in 1 month and the other patient with the same treatment but without endoscopic sphincterotomy died in 11 months.

In the 2 patients who received HAART and specific treatment for E. bieneusi and I. belli (Table) the pain resolved at 3 and 4 weeks and diarrhea at 8 and 6 weeks after initiation of HAART. In both patients with high levels of alkaline phosphatase, the levels fell to normal during therapy. The CD4 lymphocyte counts increased the basal level in both patients. The bile duct abnormality identified on ultrasonography or magnetic resonance cholangiography improved during therapy.

Our data suggest that antiretroviral therapy combined with specific pathogen therapy is a good strategy for the treatment of sclerosing cholangitis in patients with AIDS. Further investigations are necessary to confirm these preliminary observations.

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CYANOTIC CHANGE OF COLON MUCOSA IN A PATIENT WITH RAYNAUD'S **PHENOMENON**

To the Editor:

Raynaud's phenomenon is an episodic cutaneous color change in the fingers or toes associated with vasospasm, which may be precipitated by cold or emotional stimuli and which is known to be caused by various factors, including connective tissue diseases (1). In patients with connective tissue diseases, the idea that Raynaud's phenomenon may occur in the visceral organs has been suggested (2-4). We treated a patient with systemic lupus erythematosus (SLE) in whom colonic cyanosis was observed on colonoscopy simultaneously with abdominal colic pain, which may suggest Raynaud's phenomenon in the colon.

A 66-year-old woman had suffered from recurrent transient abdominal colic pain and Raynaud's phenomenon, precipitated by walking or hand washing, for the last 10 years. In September 2001, she was admitted to our hospital because of abdominal colic pain. Physical examination revealed tenderness of the midabdomen and purpura on the lower legs; transient Raynaud's phenomenon was also observed in the fingers. Hematological examination revealed a white blood cell count of $2.5 \times 10^9 / \mu L$ (51.3% segmented neutrophils, 37.5% lymphocytes). The erythrocyte sedimentation rate was 110 mm/h. Antinuclear antibody was present at a titer of >1:1280 with speckled and homogeneous patterns. Anti-double-stranded DNA antibody and anti-Ro antibody (SS-A) were also present. The CH₅₀ level had decreased to 17.1 U/mL. The results of the tests for cryoglobulin, myeloperoxidase-antineutrophil cytoplasmic antibodies (ANCA), and proteinase 3-ANCA were negative. A skin biopsy specimen obtained from the right lower leg demonstrated leukocytoclastic vasculitis. On colonoscopy, the color of the ascending colon mucosa changed from the normal color to purple immediately after the routine injection of air, indicative of colonic cyanosis (Figure 1). The cyanotic lesions appeared with random and patchy distribution, extending from the cecum to the rectum. There were neither hemorrhagic nor necrotic lesions in the mucosa of the cyanotic areas. A mucosal biopsy specimen obtained from a cyanotic area in the colon revealed no abnormal findings. Simultaneously with the observed cyanotic change, the patient had an at-