Pathology, All India Institute of Medical Sciences, New Delhi).

There are few descriptions of the changes in the duodenum and jejunum while no reports of changes due to portal hypertension (PHT) are well documented. Changes occurring in the gastric and colonic mucosa of patients with asymptomatic transaminisits: implications in diagnostic approach. J Gastroenterol Hepatol 2004;19:1291-9

The etiology of ARA T could be established in 65 (97%) patients. Non-alcoholic steatohepatitis (NASH) and chronic viral hepatitis were the etiological diagnoses in 24 (36%) patients each. Drug-induced hepatitis in 6 (9%), autoimmune hepatitis in 3 (4.5%), alcohol consumption in 2 (3%), acute anicteric hepatitis in 2 (HBV 1, HEV 1), and secondary hemosiderosis, granulomatous hepatitis, primary sclerosing cholangitis and overt cirrhosis in one patient each were the other etiologies diagnosed. Serology and biochemical tests established a definitive diagnosis in 23 (34%) patients, PCR for HBV and HCV in another 14 (21%) patients, and liver biopsy in another 28 (42%) patients.

The authors conclude that ileal varices and portal hypertensive ileopathy in patients with cirrhosis and portal hypertension. Gastrointest Endosc 2004;60:778-83

Changes occurring in the gastric and colonic mucosa due to portal hypertension (PHT) are well documented. There are few descriptions of the changes in the duodenum and jejunum while no reports of changes that occur in the ileum are available.

Forty-four patients with cirrhosis of liver and PHT (39 men; mean age 32.4 [7.7] y; Child-Pugh class A 30 patients, B 11, C 3; esophageal varices in all, portal hypertensive gastropathy [PHG] 19, gastric varices 10) and 50 control patients with suspected irritable bowel syndrome underwent terminal ileoscopy.

Ileal varices and/or portal hypertensive ileopathy (PHI) were seen in 16 patients (36%) with cirrhosis and none of the controls (p<0.05). Anorectal varices were present in 19 (43.2%) and changes of portal hypertensive colopathy (PHC) in 23 (52.3%) patients with cirrhosis. Ileal varices were observed in 8 patients (18%) and PHI in 11 patients (25%). Ileal varices were not related to Child-Pugh class, grade of esophageal varices, esophageal variceal bleeding or obliteration, PHG, gastric varices, anorectal varices, PHI or PHC. PHI correlated with the presence of PHC (9/11 patients with ileopathy had PHC). No patient had bleeding from the ileal lesions.

The authors conclude that ileal varices and mucosal changes of PHI occur in about one-third of patients with cirrhosis and PHT. Ileopathy was more common in patients who also had changes of PHC.

Patients with inflammatory bowel disease (IBD) often develop nephrolithiasis. Fat malabsorption and absence of the intestinal oxalate-degrading bacteria, Oxalobacter formigenes, may cause hyperoxaluria and calcium oxalate stone formation.

Stool samples were analyzed for the presence of O. formigenes using PCR and southern blotting in patients with IBD (n=48; ulcerative colitis [UC] 37, Crohn's 11) or renal stones (RS; n=87) and healthy controls (n=48). 24-h urinary excretion levels were analyzed spectrophotometrically in patients and in 13 controls. Five patients with IBD (10.4%; UC 3, Crohn's 2) had RS. Five (10.4%) patients with IBD, 25 (29%) with RS, and 27 (56%) healthy controls had O. formigenes colonization. Patients with IBD and RS had higher urinary oxalate levels and calcium excretion than the controls. Urinary magnesium was higher in IBD than in RS and controls. Urinary citrate was comparable in the three groups. None of the 5 patients with IBD with RS had colonization with O. formigenes. Patients without O. formigenes had higher urinary oxalates than those with it.

The authors conclude that intestinal colonization with O. formigenes is infrequent in patients with IBD and RS. This contributes to hyperoxaluria, which along with hypercalcuiuria contributes to RS in these patients.

Compiled by Sundeep Shah