

Inflammatory bowel disease presenting with nephrotic syndrome and cholestasis

Nefrotik sendrom ve kolestazla prezente inflamatuvar barsak hastalığı

To the Editor,

Crohn's disease (CD) and ulcerative colitis (UC) are the main entities of chronic inflammatory bowel diseases (IBD). Although both of them primarily involve the gastrointestinal tract, a significant percentage of patients (6% to 47%) may also have extraintestinal manifestations (1). Hepatic and renal involvement may be seen in association with IBD (2-4).

A 21-year-old male presented with a 3-week history of leg edema and abdominal distention. His laboratory tests were as follows; creatinine: 2.3 mg/dL (N: 0.7-1.3), albumin: 2.3 g/dL (N: 3.5-5.2), alanine aminotransferase: 68 IU/L (N: 0-55), aspartate aminotransferase: 135 IU/L (N: 5-35), alkaline phosphatase: 1119 IU/L (N: 40-150), gamma-glutamyl transferase: 545 IU/L (N: 9-64), and total bilirubin: 0.5 mg/dL (N: 0.2-1.3). Abdominal ultrasonography (USG) findings revealed hepatomegaly, increased echogenicity of the liver, ascites, splenomegaly, and signs of portal hypertension. There were hypoalbuminemia and massive proteinuria (20 g/24 hr). The morphological evaluation

of the renal biopsy by light microscopy exhibited adhesion between the tuft and Bowman's capsule in 3 of every 25 glomeruli, consistent with focal segmental glomerulosclerosis (FSGS) tip variant (Figure 1). The clinical pre-biopsy diagnosis of cirrhosis was based upon symptoms. The diagnostic histological features in needle biopsy specimens were enlargement of portal tracts, periportal fibrosis with minimal periportal inflammation, marked ductular reaction and extension of bridging fibrosis, progressive atrophy of bile duct epithelium, diffuse bile ductular proliferation and periductal "onion skin" fibrosis. Final diagnosis was made as primary sclerosing cholangitis (PSC) which leads to cirrhosis (Figure 2). The patient complained of abdominal pain and diarrhea, suggestive of IBD, during the follow-up. A colonoscopy with biopsy performed to evaluate the symptoms revealed normal terminal ileum, blunting of the submucosal vascular pattern, and increased mucosal granularity with edema. The histological diagnosis was IBD based on these findings, especially

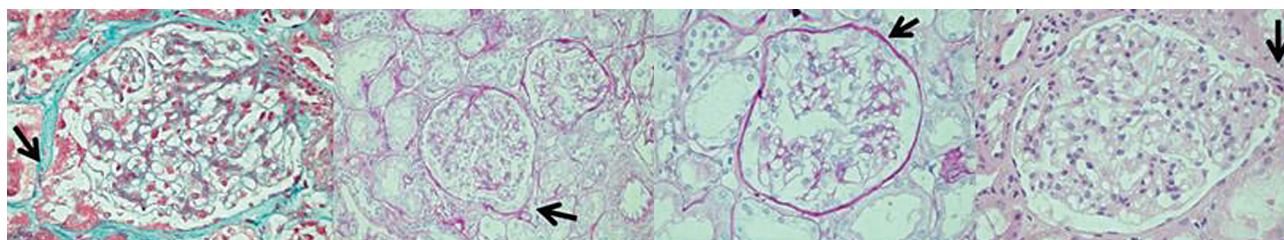


Figure 1. Masson (x40), PAS (x20), PAS (x40), HE (x40)

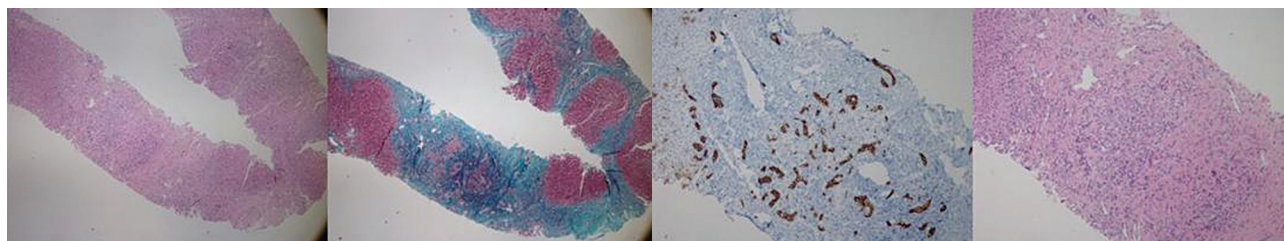


Figure 2. Brown cytokeratin (x10): ductular proliferation, Masson Trichrome (x4): fibrosis HE (x4 and x10).

Address for correspondence: Özkan GÜNGÖR

Ege University School of Medicine, Department of Nephrology,
İzmir, Turkey

Phone: + 90 232 390 42 54

E-mail: ozkangungor@yahoo.com.tr

Manuscript received: 28.02.2012 Accepted: 18.04.2012

doi: 10.4318/tjg.2013.0835

mucosal distortion (Figure 3). A combination of oral mesalazine, ursodeoxycholic acid, and prednisone treatment was started. There was no diarrhea during follow-up, proteinuria decreased, and renal function improved at third month. There were no significant changes in the liver function tests. The patient presented to emergency room with complaints of fever (39°C) and shortness of breath. His chest X-ray showed diffuse interstitial pulmonary infiltrates. The patient died from an opportunistic infection (pneumonia) ten days after hospitalization.

In conclusion, here, we reported a rare case of PSC and FSGS, which concomitantly occurred prior to IBD.

REFERENCES

1. Ardizzone S, Puttini PS, Cassinotti A, Porro GB. Extraintestinal manifestations of inflammatory bowel disease. *Dig Liv Dis* 2008; 40: 253-9.
2. Oikonomou K, Kapsoritakis A, Eleftheriadis T, et al. Renal manifestations and complications of inflammatory bowel disease. *Inflamm Bowel Dis* 2011; 17:1034-45.
3. Navaneethan U, Shen B. Hepatopancreatobiliary manifestations and complications associated with inflammatory bowel disease. *Inflamm Bowel Dis* 2010; 16:1598-619.
4. Venkatesh PG, Navaneethan U, Shen B. Hepatobiliary disorders and complications of inflammatory bowel disease. *J Dig Dis* 2011; 12:245-56.

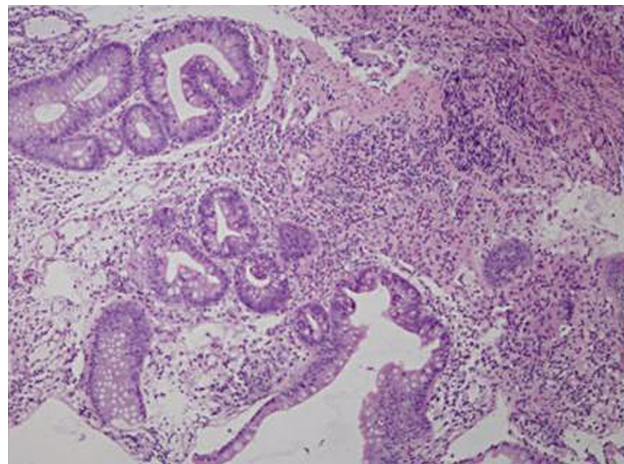


Figure 3. Intestine (x10 HE).

Özkan GÜNGÖR¹, Banu SARSIK², Erhan TATAR¹,
Ulus Salih AKARCA³, Oktay TEKEŞİN³,
Murat SEZAK², Ali BAŞCI¹

Departments of ¹Nephrology, ²Pathology and
³Gastroenterology, Ege University School of Medicine, İzmir

Pancreatic lymphoma presenting with acute pancreatitis

Akut pankreatitle prezente olan pankreas lenfoması

To the Editor,

Primary pancreatic lymphoma (PPL) is a very rare disease, accounting for less than 0.5% of pancreatic tumors (1). The clinical presentation of PPL is usually nonspecific. Most common presenting symptom reported in the literature is abdominal pain, found in about 83% of cases, followed by (in descending order of frequency) abdominal mass (58%), weight loss (50%), jaundice (37%),

acute pancreatitis (12%), small-bowel obstruction (12%), and diarrhea (12%) (2). There are few cases of PPL presenting with severe pain and acute pancreatitis in the literature (3).

A 45-year-old female presented to emergency department with worsening abdominal pain. Abdominal examination showed epigastric tenderness. The patient's laboratory findings on admission we-

Address for correspondence: Alper YAVUZ
Keçiören Training and Research Hospital,
Department of General Surgery, Ankara, Turkey
Phone: + 90 312 378 30 59
E-mail: alperavuz@hotmail.com

Manuscript received: 03.04.2012 Accepted: 04.08.2012

doi: 10.4318/tjg.2013.0597