The bladder becomes markedly over-distended from the diuretic effect of alcohol. Patients are variably obtunded, which clouds their sensory cues to void. The scenario of being awakened from sleep with sudden onset of abdominal pain supports the concept of an atraumatic rupture. In most cases the rupture is intraperitoneal, as the bladder expands at its superior portion and eventually tears at the thinnest portion, the dome.\(^1\)

Clinical features include diffuse abdominal pain and bilateral shoulder pain or hiccups due to diaphragmatic irritation. Most have inability to void and dysuria. The sudden relief of pain and distension on draining small amounts of blood-stained urine following catheterization, raises the possibility of bladder rupture.\(^3,4\) Voiding cystourethrography is the investigation of choice. Ascites:serum creatinine ratio of >1.0 is suggestive of intraperitoneal urinary leak.\(^5\) The delay in presentation and diagnosis results in significant reabsorption of urea and creatinine through the peritoneal surface and manifests as azotemia. Raised serum levels of ammonia and potassium have also been reported.\(^4\)

Surgical repair of the bladder has shown good results. Conservative management is also advocated.

The presence of high-SAAG ascites in our patient caused diagnostic confusion with underlying liver disease in a person who has a history of alcoholism.

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Received April 7, 2007. Accepted May 13, 2007

**Eosinophilic cholangiopathy – a report of two cases**

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Eosinophilic cholangiopathy is an unusual and benign form of biliary disease characterized by peripheral blood eosinophilia and cholangitis. Dramatic response to steroids is the hallmark of the disease. We present two cases of eosinophilic cholangiopathy. [Indian J Gastroenterol 2007;26:190-191]

Peripheral blood eosinophilia and eosinophilic infiltration of the gastrointestinal tract and hepatobiliary system may be seen in association with a number of conditions,\(^1-5\) but may occasionally occur in isolation. When the hepatobiliary system is involved, a radiological and clinical picture compatible with cholangitis, cholecystitis or hepatitis may be observed.

**Case 1:** A 13-year-old boy with history of bronchial asthma, on investigation for persistent fever, was diagnosed to have an abscess in the left lobe of liver with stricture of the common bile duct (CBD) on abdominal CT. This was confirmed by ERCP (Fig), during which the stricture was dilated, brushings obtained for cytology, and bile culture and stenting was done. Despite the stent, the boy had recurrent attacks of abdominal pain and fever. He was given multiple courses of antibiotics and a complete course of anti-tuberculous drugs, with no relief. Liver biopsy revealed marked eosinophilia, periporal fibrosis and bile duct damage; lymph node and bone marrow biopsy also showed plenty of eosinophilic infiltration. Peripheral eosinophilia associated tissue eosinophilia in the liver, lymph nodes and bone marrow raised the suspicion of eosinophilic cholangiopathy. Echocardiogram was normal. He was given a course of albendazole and diethylcarbamazine with no change. Subsequently, he was started on 1 mg/Kg of prednisolone.

At review after a month he was well with no symptoms. His absolute eosinophil count decreased to 400 cells/mm\(^3\) and liver function tests normalized. Repeat ERCP (Fig) showed resolution of the bile duct stricture and of the intrahepatic biliary dilatation.

**Fig:** ERCP of Case#1. ERCP at the time of diagnosis shows a long irregular stricture of common bile duct and distal common hepatic duct, with thin cystic duct and small gall-bladder (left panel). Dilatation of proximal common hepatic duct, right and left hepatic ducts, and some second order branches, is noted. Repeat ERCP after therapy with steroids (right) shows resolution of bile duct narrowing and of proximal biliary dilatation.
on 10 mg/day of prednisolone, the boy presented again with fever and severe abdominal pain. His eosinophil counts were increased and CT scan showed a para-aortic nodal mass. Ultrasound-guided aspiration was not helpful, so he underwent a diagnostic laparotomy. Ill-defined necrotic material was seen in the para-aortic region. Biopsy showed eosinophils and occasional histiocytes. He was continued on steroid therapy which was slowly tapered off over the next 6 months with good clinical response. Three months later he had a mild recurrence, which also responded to steroids, and has continued since on long-term low-dose steroids.

Case 2: A 26-year-old man presented with recurrent episodes of fever and generalized itching of four years’ duration. The fever was high grade associated with rigors, lasting for 2-3 days once in 2-3 months. This was associated with itching, but no other features of cholestasis. Laboratory evaluation revealed total leukocyte count of 11,800/mm³ (50% eosinophils). Serum bilirubin was 0.6 mg/dL, AST 23 IU/L, ALT 25 IU/L, alkaline phosphatase 424 IU/L (normal <125). Ultrasonography showed thickened wall of CBD, with an ill-defined hypechoic collar of tissue extending from the porta along the biliary radicles up to the periphery. The gall bladder wall was thickened, and a few calculi were seen. ERCP showed dilated CBD with shaggy walls along its entire length. The common hepatic duct and intrahepatic biliary radicles were dilated, with a few dilated second-order ducts. The main pancreatic duct was dilated with a few dilated side branches. Biliary sphincterotomy was done and biopsies were taken from the ampulla, which revealed significant eosinophilic infiltration. Bile cytology was negative for parasites and ova. Liver biopsy revealed moderate mixed eosinophil-rich portal and lobular inflammation. Echo-cardiogram was done to rule out cardiac involvement as a part of hypereosinophilic syndrome. Bone marrow examination revealed eosinophil-rich infiltrates but was negative for lymphoma.

With a diagnosis of eosinophilic cholangiopathy, the patient was started on prednisolone to which there was prompt symptomatic, hematological and biochemical improvement. The patient was evaluated 6 months after steroids were tapered to 15 mg/day, when the frequency of attacks had come down and repeat ERCP showed significant improvement. He was kept on a maintenance dose of 7.5 mg of prednisolone per day and remains well on follow up.

Eosinophilic cholangiopathy may coexist with pancreatitis and eosinophilic gastroenteritis. In our first case, gastric mucosal biopsy was normal; in the second case the duodenal and ampullary tissue showed eosinophilic infiltration. This makes us wonder if eosinophilic cholangiopathy is part of the spectrum of eosinophilic gastroenteritis that involves biliary and pancreatic ducts preferentially.

The criteria for hypereosinophilic syndrome (HES) – persistent eosinophilia >1500 cells/mm³ for 6 months, exclusion of other causes of eosinophilia, organ system involvement attributable to eosinophilic infiltration – were met in both the patients. Schrul et al³ reported a patient with HES, bowel disease resembling chronic inflammatory bowel disease, and cholangiopathy resembling primary sclerosing cholangitis, which responded clinically and symptomatically to hydroxyurea, the drug of choice in HES.

Obstructive jaundice in association with eosinophilic cholangitis has been reported.⁶,⁸,⁹ In all these patients the diagnosis was made after surgery for presumed malignancy. Our patient¹ also had an ill-defined mass at laparotomy and biopsy was not suggestive of any specific pathology probably because the patient was on steroids. ERCP picture suggestive of primary sclerosing cholangitis, as in our patient, has also been reported.¹

The treatment of eosinophilic cholangitis has varied from conservative watchful waiting to the use of steroids and hydroxyurea. The treatment of eosinophilic gastroenteritis has varied from short repeated courses of steroids to long-term low-dose steroid therapy or the use of steroid-sparing agents such as montelukast, ketotifen or sodium cromoglycate.⁷

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Received April 16, 2007. Received in final revised form April 30, 2007. Accepted May 13, 2007